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LOCKING THE STABLE DOOR

If someone could compile the recommendations that have been made by veterinarians to lay administrative officials, of one kind or another, during the past twenty-five years, and which have been declined or indefinitely postponed, it is believed that the total number would run well into the thousands. The kind of recommendations we have in mind includes only those that would further safeguard human and animal health and well-being. Such measures are frequently proposed to municipal officials and boards of health, county health units, state live stock sanitary boards, and occasionally to federal authorities.

It has become a part of human nature to put off locking the stable door until after the horse is stolen. An example of this sort of thing comes to mind in connection with liability insurance for veterinarians. More than once, during the past few years, we have received telegrams, special delivery and air-mail letters, from veterinarians asking for information about liability insurance. Usually the urgency of the situation has resulted from trouble—perhaps a lawsuit—and no protection.

On another page of this issue of the JOURNAL, Dr. E. E. Chase, of Portland, Oregon, reports another horse stolen because the door was not locked in time. A number of Portland citizens were stricken with trichinosis because the local meat inspection ordinance had a hole in it. Dr. Chase pointed out the hole quite

a while ago. No heed was paid to his warning. Everything was all right for a while until—suddenly—trouble—suffering—all of which was preventable. Then the hole was plugged up by amending the ordinance. So wags the world.

VETERINARY STUDENT ENROLLMENT FOR 1932-1933

More interest has been shown in the enrollment of veterinary students this fall than during any year in the last decade, or possibly longer. The principal reason for this is found in the desire of veterinary educators to observe and study the effect of lengthening the veterinary curriculum in four of our educational institutions. Iowa State College took this step a year in advance of three others (Colorado, Cornell and Kansas), which inaugurated the five-year course this year. A study of the enrollment figures brings out a number of rather interesting points.

First of all, it should be stated that comparisons of student enrollment figures, from year to year, are now somewhat complicated, as a result of four of the colleges now being on a five-year basis and nine retaining the old four-year curriculum. Even in the four institutions offering the lengthened course, we find different accounting methods used. For example, at Iowa State College, Dean Stange advises that the pre-veterinary students are called "first-year students" and those whom we used to call "freshmen" are no longer designated as such, but are called "second-year students," those whom we would call "sophomores" are called "third-year students," and so on.

At Cornell University, according to Dean Hagan, there is no pre-veterinary year. Students do not register in the veterinary course until they have completed one year of collegiate work. Dean Hagan states that there is no means of knowing how many students are preparing to enter the New York State Veterinary College, until they are actually enrolled as freshmen. One year ago there were 70 freshmen enrolled at Cornell; this year there are 27.

At Kansas State College, pre-veterinary students are enrolled in the Division of Veterinary Medicine and are regarded as veterinary students. Dean Dykstra points out that these students are taking the first year of a five-year curriculum and, for that reason, should be counted as veterinary students. If we correctly understand the situation at Manhattan, students now enrolled in the Division of General Science may start the study of veterinary medicine next year as second-year students,

without having taken the pre-veterinary studies as such, but provided they have the proper credits. Dean Dykstra reports 12 freshmen and 15 pre-veterinary students, a total of 27, against 56 freshmen in 1931.

At the Colorado Agricultural College, it is stipulated in the catalog that should the pre-veterinary year be taken at that institution the students may conveniently enroll in either agricultural or forestry courses. Dean Glover reports 23 pre-veterinary students, and only nine freshmen. This very vividly shows the effect of the new matriculation requirements at Fort Collins.

In the tabulation this year all pre-veterinary students reported are being accounted for as "special students." Of the 68 included under this head, 56 are taking the first year of the five-year course in three of the colleges. The 12 others are students with various credits and incapable of classification as freshmen, sophomores, juniors or seniors. Last year Dean Stange reported 34 pre-veterinary students enrolled in the Division of Industrial Science at Iowa State College and approximately 25 others enrolled in other divisions for pre-veterinary work. The report indicates that only 28 of these are enrolled at Ames as second-year students this fall.

The comment has been heard that if only a part of the colleges go to the five-year course, and the others stick to the four-year course, the latter will probably enroll most of the students declined by the five-year schools and desiring to finish the course in four years. Last year there were 453 freshmen and 59 pre-veterinary students, or a total of 512 new students, reported in all colleges. This year there are 383 freshmen and 56 pre-veterinary students, or a total of 439 new students (no pre-veterinary students reported by Cornell). This looks like a decrease of 73 new students from last year. The only colleges to report fewer students enrolled this year than last are five-year schools. The fourth (Kansas) shows an increase of two.

The nine colleges still on a four-year basis reported 267 freshmen in 1931. This year these same colleges report 307, an increase of 40 over last year. These figures very strongly suggest that many prospective veterinary students are not "sold" on the advantages of the five-year curriculum and are content to take the four-year course. This year Alabama reports five more freshmen than last year; Montreal, 4; Ohio, 13; Ontario, 10; Pennsylvania, 3; Texas, 2; Washington, 7. With their courses remaining the same, two colleges (Georgia and Michigan) report fewer freshmen this year than last.

The number of sophomores in college this year is about the same as last year, and the same thing holds true of the seniors. However, there are 83 more juniors this year than last. There appears to have been a considerable mortality among the 453 freshmen enrolled last year, as only 391 sophomores are reported this year. Of 388 sophomores last year, 353 returned as juniors this fall; and of 270 juniors in college last year, all appear to be accounted for, as well as four additional (possibly classed as special students last year).

Graduate studies in veterinary medicine continue to attract more students. This is reflected in the 38 graduate students reported in six of the colleges this year, compared with 30 for last year.

The accompanying table shows the detailed reports of the 1932-1933 enrollment.

Veterinary student enrollment for the college year 1932-33.

	Fr.	Soph.	Jun.	Sen.	Spec.	Grd.	Tot.	1931–32	Chg.
Alabama P. I.	24	18	19	10	0	1	72	58	+14
Colorado A. C.	9	38	23	25	23*	0	118	134	−16
Cornell Univ.	27	66	51	26	0	11	181	204	−23
Georgia S. C.	12	10	10	11	0	0	43	41	+ 2
Iowa State Coll.	28	16	64	42	18*	7	175	179	− 4
Kansas S. C.	12	49	40	45	18†	3	167	165	+ 2
Michigan S. C.	29	26	16	10	0	6	87	70	+17
Montréal, Univ. of.	16	11	12	9	0	0	48	34	+14
Ohio State U.	78	49	38	37	0	10	212	179	+33
Ontario V. C.	52	38	31	18	0	0	139	115	+24
Penna., Univ. of.	50	38	28	23	9	0	148	134	+14
Texas A. & M. C.	13	11	6	5	0	0	35	32	+ 3
Washington, S. C. of	33	21	15	13	0	0	82	73	+ 9
Totals (1932-33).	383	391	353	274	68	38	1507	1418	+89
Last year.	453	388	270	267	10	30	1418	1246	+172

*Pre-veterinary students.
†Includes 15 pre-veterinary students.

AN EXPERIMENT IN VETERINARY EDUCATION

The Board of Trustees of the University of Pennsylvania, some months ago, approved a resolution, adopted by the faculty of the Veterinary School, providing that each freshman class, beginning with the year 1932-33, should be limited to fifty selected students.

In commenting on this important step in veterinary education, Dean Dick stated that more than fifty men in a class could not

be accommodated with any degree of efficiency and the indications were that this limit will be exceeded right along unless steps were taken to limit the number of entering students. Furthermore, according to Dean Dick, the possession of a high school diploma is not *per se* sufficient qualification for starting the study of veterinary medicine. Many high-school graduates have considerable difficulty with their mid-year examinations the first year, and those who barely pass usually have trouble throughout the rest of the course.

Those institutions which have already adopted the five-year course have found substantially the same condition, and the insertion of the pre-veterinary year in between the completion of the high school course and the starting of the veterinary course proper is a means to the same end—better-trained veterinarians. The Pennsylvania experiment will undoubtedly be watched with considerable interest. By having each class composed of carefully selected students—those who will be able to carry successfully the present heavy load of the veterinary curriculum crowded into four years—the disadvantage of the extra year may be overcome.

The plan inaugurated at Pennsylvania really goes further, however. An attempt will actually be made to keep out of the veterinary profession those individuals who do not show indications of being adapted for it, in the way of personality, mentality, and so forth. It gains the institution nothing, and there is no profit to the individual, if he takes up a calling for which he later finds himself poorly adapted. Providing that suitable means are found available for judging and selecting good veterinary students, and turning away those believed to be unqualified, the latter are really being done a favor. They are being saved a lot of time, expense and effort, that otherwise might be wasted.

Dean Dick comments further as follows:

> By adopting a system of selection we hope to select men who have the best grades in high school and who, in the opinion of their teachers in science, are best adapted to veterinary medicine. In addition to this we will have a personal interview with each candidate, if possible; if not possible, we will insist on having a photograph. It is our thought at the present time that * * * we will have the Psychology Department examine each student after he has enrolled, with the view to ascertaining whether or not this service will assist us in selecting our men in the future. This in a nutshell is our plan of se'ecting students.

There are some who have expressed doubts as to the wisdom of extending the veterinary curriculum to five years. The rea-

sons given, in the main, have been largely economic ones. Stated very bluntly, these reasons amount to saying that the financial returns from the veterinary profession do not warrant spending five years in college. This is largely a matter of personal opinion, however. Everything considered, we believe that the average veterinary student will be a better veterinarian at the end of five years than he will be at the end of four years of training. Whether the extra year may better be spent in pre-veterinary studies or in post-graduate work is another question.

It is our understanding that the idea of going to the five-year course at Pennsylvania has not been given up definitely, but is being studied by a committee appointed for the purpose.

EXECUTIVE BOARD ELECTION

The polls for the primary election being held in Executive Board District 8 will be closed when the last mail is received on November 7. This date bears no relation to the presidential election scheduled for the following day, but conforms to the provisions of the by-laws for keeping the polls open for 60 days. Right here it might be said that this period is much too long except for District 7, which embraces the Philippine Islands. The experience of conducting twenty Executive Board elections during the past ten years has shown that 90 per cent or more of the ballots are cast during the first thirty days of the election. In the present case 95.3 per cent of the ballots returned by October 25 were received within thirty days of the opening of the polls.

In at least two of the five states in District 8, organized efforts are being put forth by admiring friends to elect prominent practitioners. These campaigns have injected considerable interest into the election. We have never felt that it was exactly the right spirit to get behind a candidate just because he was a practitioner, or a Bureau veterinarian, or a state official, or a college professor. On the other hand, the proper yardsticks for measuring the qualifications of candidates for election to the Executive Board are ability and willingness to serve the members in their respective districts. Moreover, experience has demonstrated that when these standards are employed, usually the best available men are selected. As now constituted, the Board is made up of four members connected with colleges, three practitioners, two federal veterinarians, two others engaged in commercial work, one state veterinarian and one county official.

HOW DO THEY GET AWAY WITH IT?

In the October issue of the JOURNAL, attention was directed to a flagrant case of meddling by a county agent, under the heading, "Why Do They Do It?" One member commented to the effect that the editorial was very timely, coming so soon after the Atlanta convention, where several hundred of our members were told inferentially by a federal official that nothing of the kind was tolerated by the Extension Service. This member suggested that our editorial should have been captioned, "How Do They Get Away with It?"

Another member very kindly sent a newspaper clipping containing a letter written by a county agent in reply to a request that he come out and see a sick horse. Realizing his limitations and at the same time remembering what are and what are not proper functions of county agents, this level-headed gentleman wrote as follows:

> *Remember* that I am *no veterinarian* and have no right to treat sick animals even if I knew how. A registered veterinarian is always the best bet when an animal is sick. Many will call me, for there is no charge connected with my calls. Others will call a local *so-called doctor*, who has not had any training except what he has picked up on treating your animals, sometimes with good results and sometimes with death as the result. One such case came to my notice last Wednesday and the owner will be out a good work horse as well as a young colt as the result. He finally called a veterinarian after the horse had been sick a week with only the *so-called doctor* present. These so-called doctors cannot charge for their work, the law states, but you pay for their gasoline and time, etc., and many times this charge is small compared to the loss of the animal that may result. If the animal is not sick, leave it alone and do not give it medicine, but if sick, get a man that has proved to the state at large, as well as his neighbors, that he is competent to treat all manner of cases.
>
> There have been some men castrating colts locally that have charged more than a local veterinarian for the job and more deaths of such colts have resulted than the local veterinarian has ever had. Not one case has been lost this spring by the local veterinarian, while there are many such deaths that can be traced to these other men.

Incidentally it might be stated that the writer of this letter was largely responsible for the veterinarian (mentioned in the letter) locating in the county. All of this happened in a state where veterinarians and county agents have been "getting along" together with very little trouble.

For about a day it looked like a tie score at 1 to 1, but it did not stay that way very long. At Cedar Rapids, Iowa, we had a conference with Dr. H. E. Curry, chairman of the A. V. M. A. Special Committee on Agricultural Extension Service. He has

very diligently taken hold of his new job and is collecting "facts" and other evidence. Here is a sample, being the quarterly report "respectfully submitted" by County Agent P. H. Ward, of Thomasville, Ga., to the Commissioners of Thomas County, on September 10, 1932, just about two weeks after the Atlanta convention:

Gentlemen:

For your information, I beg to submit the following report: During the months of June, July and August, I traveled on official duty 4,695 miles, visited 225 farms on official duty. Held 71 personal consultations and answered 154 phone calls.

Treated 2,764 head of hogs to prevent cholera. Held one cooperative hog sale, selling 16,210 pounds, bringing $636.44. Wrote two news articles and 95 official letters. Distributed 80 agricultural bulletins.

Treated 303 head of hogs for swine plague, 149 head of hogs for internal parasites, and 108 head of cows. Furnished plans to 3 farmers for meat curing houses and 7 plans for corn weevil proof barns.

Made one tour with a specialist from the State College of Agriculture. Spent June 27-30th at Athens, Ga., with club boys and attending a state conference of county agents.

Here is a county agent who modestly reports having vaccinated 2,764 hogs against cholera, treated 303 more for swine plague and 149 for internal parasites, over a three-month period. His cattle practice was not so bad, either. He treated 108 head of cows, but he does not say for what. Possibly the ailments were too numerous to mention in such a brief report. Our records show that there are at least two graduate veterinarians located in Thomas County. Well, perhaps it *is* time to ask, "How Do They Get Away with It?" Perhaps Dr. Warburton can answer the question.

REMEDY BUSINESS NOT SO GOOD

A report recently reached us, from a source believed to be quite reliable, to the effect that the manufacturers of a new remedy for a certain animal disease had spent a large amount of money advertising the nostrum to the laity in several periodicals. The results were very disappointing—to the nostrum vendors. When they counted up the cost of the advertising, they found that each lead had cost them about sixteen dollars. Only two leads out of every five (40 per cent) resulted in actual sales. If our calculations are correct, it cost about forty dollars in advertising to make one sale.

There is a strong suggestion here that owners of animals are beginning to heed the warnings of the U. S. Food and Drug

Administration, against purchasing many of the so-called "cures" which have flooded the market in the past. Then, too, credit should be given the A. V. M. A. Committee on Proprietary Pharmaceuticals, for bringing about a better state of affairs as far as the advertising pages of many of our farm papers are concerned. As matters now stand, manufacturers of remedies of questionable merit find it practically impossible to purchase advertising space in the better publications, as a direct result of the strict censorship now imposed on this kind of advertising. Let the good work continue.

APPLICATIONS FOR MEMBERSHIP

(See July, 1932, JOURNAL)

FIRST LISTING

KESTER, WAYNE O. 50 E. Buchtel Ave., Akron, Ohio.
D. V. M., Kansas State College, 1931
Vouchers: C. H. Case and J. F. Planz.

SKOOG, HARRY E. c/o Y. M. C. A., Pittston, Pa.
D. V. M., Kansas State College, 1930
Vouchers: J. J. Martin and R. R. Dykstra.

STEINMAN, FRANK C. 303 W. T. Waggoner Bldg., Fort Worth. Texas.
D. V. M., Agricultural and Mechanical College of Texas, 1929
Vouchers: J. J. Reid and N. F. Williams.

TERRY, EDWARD E. 3606 Welsh Road, Holmesburg. Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1932
Vouchers: M. A. Emmerson and E. J. Marshall.

Applications Pending

SECOND LISTING

(See October, 1932, JOURNAL)

Baird, John, Coldstream Stud, Lexington, Ky.
Chase, Chas. M., 3264 Utica St., Denver, Colo.
Ferguson, Howard F., 15 Harrington St., Newport, R. I.
Karr, James R., Coshocton, Ohio.
Miller, Carroll L., 240 Madison St., Oak Park, Ill.

The amount which should accompany an application filed this month is $5.83, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

STATE BOARD EXAMINATION

Nebraska Bureau of Examining Boards. State House, Lincoln, Neb. State veterinary examination, November 15-16, 1932. Applications must be on file at Bureau 15 days prior to examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Neb.

COMING VETERINARY MEETINGS

Central New York Veterinary Medical Association. Onondaga Hotel, Syracuse, N. Y. November 1, 1932. Dr. W. B. Switzer, Secretary, R. 5, Oswego, N. Y.

Connecticut Veterinary Medical Association. Bridgeport, Conn. November 2, 1932. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. & 103rd St., New York, N. Y. November 2, 1932. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. November 2, 1932. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. November 8, 1932. Dr. E. E. Sweebe, Secretary, 14th St. & Sheridan Road, North Chicago, Ill.

Hudson Valley Veterinary Medical Society. Poughkeepsie, N. Y. November 9, 1932. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. November 9, 1932. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Michigan-Ohio Veterinary Medical Association. Adrian, Mich. November 10, 1932. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Tulsa County Veterinary Association. Tulsa, Okla. November 10, 1932. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Interstate Veterinary Medical Association. Elks Bldg., Omaha, Neb. November 14, 1932. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.

Polk County Veterinary Medical Association. Des Moines, Iowa. November 14, 1932. Dr. A. H. Quin, Jr., Secretary, 313 49th St., Des Moines, Iowa.

New England Veterinary Medical Association. Hotel Bradford, Boston, Mass. November 14-15, 1932. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. November 15, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. November 16, 1932. Dr. E. E. Jones, Secretary, 1451 Mirasol St., Los Angeles, Calif.

Keystone Veterinary Medical Association. Philadelphia, Pa. November 23, 1932. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

United States Live Stock Sanitary Association. Hotel LaSalle, Chicago, Ill. November 30, Dec. 1-2, 1932. Dr. O. E. Dyson, Secretary, 45 Live Stock Exchange, Wichita, Kan.

Horse Association of America. Blackstone Hotel, Chicago, Ill. November 30, 1932. Wayne Dinsmore, Secretary, Union Stock Yards, Chicago, Ill.

National Association of B. A. I. Veterinarians. Hotel LaSalle, Chicago, Ill. December 1, 1932. Dr. F. A. Imler, Secretary, 19 Federal Bldg., Kansas City, Mo.

Maryland State Veterinary Medical Association. Medical Hall, 1211 Cathedral St., Baltimore, Md. December 8, 1932. Dr. E. M. Pickens, Secretary, College Park, Md.

Southeast Kansas Veterinary Medical Society. Oswego, Kan. December 9, 1932. Dr. L. F. Barthelme, Secretary, Parsons, Kan.

Nebraska State Veterinary Medical Association. Yancey Hotel, Grand Island, Neb. December 13-14, 1932. Dr. E. C. Jones, Secretary, c/o Platte Valley Serum Co., Grand Island, Neb.

American Association for the Advancement of Science. Atlantic City, N. J. December 27-31, 1932. Dr. Chas. F. Roos, Secretary, Smithsonian Institution Bldg., Washington, D. C.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 4-5, 1933. Dean G. A. Dick, Secretary, 39th St. and Woodland Ave., Philadelphia, Pa.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 9-10, 1933. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.

Ohio State Veterinary Medical Association. Neil House, Columbus, Ohio. January 11-12, 1933. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Cornell University, Annual Conference for Veterinarians at. Cornell University, Ithaca, N. Y. January 12-13, 1933. Dean W. A. Hagan, Secretary, New York State Veterinary College at Cornell University, Ithaca, N. Y.

ANEMIA IN RELATION TO VACCINATION SHOCK IN YOUNG PIGS*

By C. N. McBRYDE, *Ames, Iowa*

Field Station, Biochemic Division, U. S. Bureau of Animal Industry

INTRODUCTORY

So-called "vaccination shock" is a more or less severe reaction which sometimes occurs in young pigs following the injection of anti-hog cholera serum and is a phenomenon or occurrence that seems to have attracted attention only within the past few years.

Since 1929, cases of vaccination shock have been reported from time to time by practicing veterinarians and the occurrence of severe, shock-like reactions following the immunization of young pigs has not infrequently proven quite alarming to veterinarians and their clients. The phenomenon has also become a source of worry to some serum-producers.

Two facts in connection with vaccination shock were soon established. The phenomenon was found to be confined chiefly to young pigs weighing under 40 pounds, and seemed to be associated with the injection of *clear, heated* serum.

Various theories have been advanced to explain the phenomenon. Within the last few years, the intraperitoneal method of administering serum has been adopted by many veterinarians, especially for the immunization of young pigs. When vaccination shock was first observed, it was thought that the injection of cold serum might cause the shock. It later developed, however, that shock occurred irrespective of the temperature of the serum. It was then found that shock was more apt to occur and the reaction be more severe when serum is injected intraperitoneally instead of subcutaneously, which led a good many veterinarians to abandon this method of administering serum. Some veterinarians thought that vaccination shock was a question of feed, and regarded shock as an anaphylactic reaction. It was thought that the simple expedient of depriving the animals of feed for 24 hours would serve to eliminate shock. The latter precaution undoubtedly does serve to lessen the severity of some symptoms, more especially the nausea and vomiting, but does not suffice to eliminate shock.

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*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

The first scientific investigation of vaccination shock was made by Munce and Hoffman,[1] who carried out an extensive and well-planned series of experiments designed to determine the nature and cause of this puzzling, and until then little understood, phenomenon. The main points brought out by these investigators were as follows:

(1) It was possible to produce experimentally a condition in a large number of young pigs which to all appearances is the same as, or at least very similar to, vaccination shock sometimes observed in the field following the vaccination of young pigs.

(2) This condition was produced experimentally by the injection of * * * * clear serum heated at certain temperatures in the process of its preparation * * * *

(3) The condition occurred most regularly in the intraperitoneal injections of pigs when the serum injected was heated for thirty minutes at temperatures ranging from 60 to 62° C., however, reactions also followed the injection of serum heated for the same period at a temperature as low as 58° C. and as high as 64 to 65° C. * * * *

(4) There is apparently a great variation in the susceptibility of pigs to the shock-inducing properties of heated clear serum. This susceptibility is apparently greater in suckling pigs than in pigs which have been weaned. Experimental work referred to would indicate that this susceptibility ceases at some age in practically all pigs. The age at which such immunity develops, however, has not yet been determined.

(5) No fatalities occurred as a result of reactions reported in these experiments. * * * * This is in accord with field reports which indicate a very low mortality for this condition.

A rather extensive study of vaccination shock was made at the Field Station of the Biochemic Division of the Bureau of Animal Industry at Ames, during the summer of 1931. The results of this study served to confirm the findings of Munce and Hoffman,[1] except that no shock was noted with serum heated at 58° C. for 30 minutes. Chemical studies, which were carried out by Mr. W. C. Powick, in connection with this work, indicated rather clearly that the shock-producing material in the serum is precipitated by ammonium sulfate in the concentrations usually employed to precipitate the euglobulins.

In connection with the Bureau experiments at Ames, which were carried out for the most part on farms, with young pigs weighing from 8 to 45 pounds, it was very evident that young suckling pigs were more susceptible than older weaned pigs. A considerable variation also was noted in individual pigs and in groups of pigs of approximately the same age, and this matter of susceptibility to shock proved a very puzzling one. No satisfactory explanation could be offered to explain it when a brief summary of the experiments was published in the annual report of the Chief of Bureau.[2]

In a second article on vaccination shock in young pigs, Munce[3] made the following statement:

> Our experimental work does not indicate that the use of a heated clear serum is the fundamental cause of the reactions because a number of young pigs and entire groups of them are encountered which do not manifest any appreciable susceptibility to the heated product. In other words, the fundamental cause of these reactions concerns some factor or factors which render some pigs SUSCEPTIBLE to the shock-inducing property of heated clear serum. A great many possible factors have been considered in this connection. The cause of this susceptibility may eventually be traced to some article in the diet of the pig or the sow. To date, efforts to produce susceptibility to shock by means of the diet of the pig have been unsuccessful.

In the article just referred to, Munce states that observations and reports indicated that spring pigs were more susceptible to shock than fall pigs, which he thought might point to some deficiency in the diet of the sow, this assumption being based on the fact that the diet of the sow during the spring gestation period is lacking in the fresh vegetation usually included in the diet prior to fall farrowing.

A review of the literature has failed to reveal any satisfactory explanation of the individual and group susceptibility of certain pigs to vaccination shock. As already mentioned, this had proved to be a very puzzling matter during the course of the experiments carried out at the Field Station of the Biochemic Division of the Bureau at Ames, in the summer of 1931, but there had been no opportunity to investigate this point further and the matter had been allowed to rest.

Present Investigation

In the course of some investigations at a large garbage-feeding ranch in southern California, in the winter of 1931-1932, an excellent opportunity was afforded to make further observations and studies on vaccination shock in young pigs. On this ranch, hundreds of young pigs were being farrowed continually. Many of the dams with their litters were being held in small pens floored with concrete until weaning time. When little pigs held under these conditions were vaccinated around five weeks of age, very severe reactions occurred, which often terminated fatally. An occurrence of this kind was noted in a group of 191 pigs which were vaccinated in the afternoon. On the following morning 15 of these pigs were found dead. Most of these pigs probably died within a short time after vaccination, although they were not observed continuously following vaccination. It was very evident, however, that the death loss was due to vac-

cination shock, as one pig had succumbed before the entire lot had been vaccinated.

The mortality of nearly 8 per cent was unusually high for vaccination shock. These pigs were from 28 sows, having an average of a little better than 6½ pigs to the litter. They were confined in two rows of individual pens. There were 14 adjoining pens in each of the two rows of pens. Each pen consisted of an enclosure 7 by 24 feet, with a house 7 by 8 feet occupying one end and opening on a concrete yard 7 by 16 feet. The houses in one group of pens had wooden floors, while those in the other group had concrete floors. The houses, which were partly open in front, faced to the west and the paved yards or runways of the individual pens were separated by board fences. The pigs had been confined in these pens since birth and were about five weeks of age at the time of vaccination, which was at the end of January. The daily ration consisted of two feedings, one of garbage and the other of grain (a barley mixture). The dead pigs, which had lain overnight, were in good condition for autopsy, as the weather was quite cold at the time. Postmortem examination of the pigs revealed a pronounced anemia. The subcutaneous and muscular tissues were very pale and pallor was noted in the internal organs, especially the liver and kidney.

The fact that these pigs suffered from anemia was not surprising, as the conditions under which they were held were very favorable to the development of this condition. The weather had been rather cold, with spells of rain, and the sows and little pigs naturally would not sleep outside on the cold concrete but remained in their houses a good part of the time. The little pigs were thus deprived of sunlight, for when they went outside in the yards the sunlight was cut off to a large extent by the east and west partition fences. Owing to the limited size of yards, there was little opportunity for exercise and the little pigs were thus deprived to a large degree of both sunshine and exercise and had no access to soil. The death losses, as a rule, occurred here and there in different litters, but in one litter of six there was a death loss of five. The death loss was practically the same in the two groups of pens. In the 14 pens which had houses provided with wooden floors, seven pigs died, while in the 14 pens with houses having concrete floors, eight pigs succumbed.

In view of the fact that experiments carried out at the Bureau Station at Ames, with many different lots of serum, carefully

heated at temperatures ranging from 58 to 62° C., had shown that the injection of serum heated much above 58° C. was apt to cause shock in young pigs, the first thought was that the serum might possibly have been somewhat overheated in process of preparation. Fortunately, however, there was a very good check on the serum, for the ranch veterinarian had used the same serum only a day or two previously for the vaccination of 84 litters (542 pigs) about five weeks of age, which had been kept in large brooder pasture pens, where they had ample room for exercise and access to soil and sunlight. There was no loss from shock in these pigs, which were of the same approximate age and received the same dosage of serum as the pigs confined in the individual pens, where the heavy death loss evidently was due to some condition in these pigs and was not the fault of the serum.

In addition to the check on the serum that has just been cited, complete data nevertheless were obtained through Government channels in regard to the preparation and testing of the particular lot of serum used in this instance. The report showed that the serum had not been overheated in the course of preparation, that it had proved satisfactory for potency and purity and no shock-like reaction had been noted in connection with the usual Bureau-supervised tests made prior to the release of this serum.

As it seemed quite probable from the postmortem examinations that the high mortality from vaccination shock in the case of the pigs confined in the individual pens was associated with an anemic condition, experiments were arranged in coöperation with the ranch veterinarian with a view to throwing more light on the relation of anemia to vaccination shock.

In order to afford a better understanding of the experiments to be described later, it seems desirable first to give a brief description of the methods of handling suckling pigs at the ranch and review briefly the literature on the subject of anemia in young pigs.

Methods of Handling Suckling Pigs at Large Garbage-Feeding Ranch in Southern California

The ranch has two brooder units, each of which has twelve groups of farrowing-pens, known as "batteries." Each battery consists of 25 farrowing-pens. Thus there are 300 farrowing-pens in each brooder unit and the two units therefore will accommodate 600 sows.

The farrowing-pens are 7 x 26 feet, with a farrowing-house 7 x 8 feet. The farrowing-houses, which extend across the pens,

are provided with concrete yards at front and back, one yard being 7 x 10 feet and the other 7 x 8 feet. The feeding is done in the smaller yard and a water-trough is also placed in this yard. The houses are provided with farrowing-rails and the bedding consists of wood shavings. Each sow has an individual farrowing-pen and is kept in this pen with her litter until the pigs are approximately three weeks of age. The sows and litters are then transferred to "brooder pasture pens," which are dirt inclosures, or dry lots, 48 x 100 feet, provided with open shelter houses, drinking-fountains, and concrete "bath tubs" or wallows. Six sows with their litters are placed in each brooder pasture pen and the pigs remain in these pens until weaning time, when they are transferred to "weaner pasture pens" and are known as "weaners." During this time, that is, from farrowing to weaning time, the sows and pigs are fed garbage once a day and grain (a barley mixture) once a day.

In connection with each of the brooder units are groups of the individual pens, which already have been described. When the ranch, which handles around 50,000 hogs a year, is operating at full capacity, that is, producing its full quota of baby pigs, the individual pens are used to supplement the brooder pasture pens, but the great majority of the young pigs pass through the latter, which is a fortunate thing, as will be shown later.

Anemia in Young Pigs

Anemia in young pigs has received considerable attention from research workers in animal diseases within the past few years and three factors are commonly supposed to play a part in its causation. These factors are lack of sunshine, lack of exercise, and lack of contact with soil or mineral matter.

In veterinary practice in the Middle West, anemia is encountered chiefly in the early spring pigs, which have been more or less confined indoors and thus subjected to all three of the causative factors which have just been mentioned.

Braasch,[4] in Schleswig-Holstein, in 1890, was one of the first to report on anemia in young pigs. He states that the death losses from anemia among sucklings and young pigs were so heavy that swine production had to be abandoned on some farms. He noted that the disease became more prevalent after the more modern types of hog-houses came into use. In his opinion, anemia was a result of the improper handling of the breeding animals.

M'Gowan and Crichton,[5] in England, in 1923, described quite accurately the gross and microscopic lesions seen in anemia.

They believed that anemia resulted from a deficiency of iron in the ration and that it could be prevented by the addition of iron oxid. They believed that anemia was identical with so-called cottonseed poisoning in pigs and thought it probable that enzoötic hepatitis was due to anemia.

Doyle,[6] at Purdue University, has made extensive studies of anemia in young pigs. He is of the opinion that lack of contact with soil is the chief factor in the causation of anemia. He considers that access to blue-grass sod within a week after birth will protect young pigs against anemia and that access to soil alone also provides a considerable degree of protection. For the prevention of anemia in young pigs confined in barns or in concrete yards, he suggests the simple expedient of throwing chunks of sod in the pens where the pigs are confined. Doyle emphasizes the great importance of preventing anemia in young pigs in the following statement:

> If the disease once develops, it may cause serious death losses and also do lasting damage to the pigs which survive.

He also states:

> It appears quite certain that the disease accounts for a large portion of the disappointing results which follow attempts to raise pigs in barns and central hog-houses.

Hastings[7] regards lack of exercise and overfeeding as the chief factors in the causation of anemia.

Fulton,[8] at the University of Saskatchewan, agrees with Doyle in regarding lack of contact with soil as the chief factor in the anemia of young pigs, but also calls attention to the value of exercise in the prevention of this condition.

With the double object in view of obtaining more definite data on the relation of anemia to vaccination shock and at the same time throwing some additional light on the relative importance of the factors which appear to be concerned in the production of anemia, experiments were carried out with young pigs held under varying conditions.

Experiments to Determine Whether Anemia Is a Factor in Vaccination Shock and the Relative Importance of the Factors Concerned in the Production of Anemia

In one experiment there were three groups of pigs held under varying conditions, as follows:

Group 1: Fourteen sows with their litters (94 pigs) were transferred on February 6, from the farrowing-pens, at the usual age, to individual pens facing west and similar to those in which the heavy loss from vaccination shock had occurred,

as previously described. Each sow and her litter occupied an individual pen. The houses in these pens had wooden floors, while the yards were concrete. The yards were separated by board fences, which cut off the sun from the south to a large extent. The pigs were from 18 to 22 days old when moved from the farrowing-pens to the individual pens, the majority being from 19 to 21 days of age, or around 3 weeks. The sows and pigs were placed in the individual pens on February 6 and were fed in the usual manner, with alternate feedings of garbage and grain ration.

On March 1, when the pigs had been in the pens for 23 days and were then about 6 weeks of age, hemoglobin tests were made on all of the pigs in each of the fourteen litters, the pigs in each litter being marked with green paint for subsequent identification. The hemoglobin determinations were made with the original Tallquist scale.

On the following day (March 2), the pigs were given serum-simultaneous treatment with clear, heated serum. The serum was given subcutaneously in the groin and the virus was injected in the subcutaneous tissues over the ham. The pigs weighed from 20 to 30 pounds, with an average of 25 pounds, and received approximately 1 cc of serum per pound.

The results of the hemoglobin tests and the occurrence of shock following vaccination are shown in tables I and II.

An analysis of the results following vaccination in group 1, which consisted of 94 suckling pigs, about 6 weeks of age, confined in concrete yards since birth, with no access to soil and a limited amount of sunshine, shows that 38 of these pigs (about 40 per cent) suffered from vaccination shock; there were 14 cases of severe shock and 7 deaths from shock. In the following summary, the pigs are grouped according to the extent or degree of shock following simultaneous treatment, with the hemoglobin range shown for each group:

56 pigs (hem. 45-70%)—no noticeable shock
4 pigs (hem. 45-65%)—mild shock
20 pigs (hem. 30-65%)—pronounced shock
14 pigs (hem. 25-55%)—severe shock

In this group of 94 pigs, thirty-eight (40.4 per cent) showed symptoms of shock, which was severe in fourteen (nearly 15 per cent). Seven pigs (7.4 per cent) died from shock, which was a high mortality compared with losses in the field, where the mortality is very low, seldom more than 1 or 2 per cent, and frequently none at all. In the case of the fourteen pigs which suffered from severe shock, the hemoglobin readings for the indi-

TABLE I—*Percentage of hemoglobin and degree of shock following simultaneous treatment shown by 94 suckling pigs (group 1), about 6 weeks of age, confined in small concrete yards facing west. These pigs had no access to soil, comparatively little sunlight and limited opportunity for exercise.*

LITTER	PIGS	NO.	HEMOGLOBIN (%)	SHOCK	REMARKS
1	6	1	50	Pronounced	
		2	60	None	
		3	60	None	
		4	30	Pronounced	
		5	30	Severe	Recovered
		6	30	Severe	Died
2	7	1	50	None	
		2	60	None	
		3	50	Pronounced	
		4	70	None	
		5	60	None	
		6	45	None	
		7	55	None	
3	6	1	55	None	
		2	60	None	
		3	45	Pronounced	
		4	55	None	
		5	60	None	
		6	55	None	
4	7	1	60	None	
		2	60	None	
		3	55	None	
		4	55	None	
		5	60	None	
		6	55	None	
		7	30	Pronounced	
5	5	1	60	None	
		2	55	None	
		3	45	None	
		4	55	Mild	
		5	50	None	
6	10	1	55	None	
		2	60	None	
		3	70	None	
		4	60	None	
		5	60	None	
		6	70	None	
		7	65	None	
		8	55	None	
		9	55	None	
		10	60	None	
7	8	1	60	None	
		2	45	Mild	
		3	35	Pronounced	
		4	40	Pronounced	
		5	30	Pronounced	
		6	35	Pronounced	
		7	45	None	
		8	45	None	

TABLE I—(*Concluded*).

LITTER	PIGS	NO.	HEMOGLOBIN (%)	SHOCK	REMARKS
8	5	1	65	None	
		2	55	None	
		3	55	Severe	Recovered
		4	35	Pronounced	
		5	25	Severe	Died
9	7	1	35	Severe	Recovered
		2	65	Mild	
		3	45	Pronounced	
		4	45	None	
		5	55	Pronounced	
		6	45	None	
		7	30	Severe	Died
10	8	1	70	None	
		2	60	Pronounced	
		3	60	Pronounced	
		4	65	None	
		5	60	None	
		6	70	Pronounced	
		7	70	Pronounced	
		8	35	Severe	Died
11	4	1	45	Severe	Recovered
		2	45	Pronounced	
		3	45	Severe	Died
		4	55	Severe	Recovered
12	7	1	55	Severe	Recovered
		2	55	None	
		3	55	None	
		4	45	Mild	
		5	60	Pronounced	
		6	60	None	
		7	55	Severe	Recovered
13	6	1	55	None	
		2	60	Pronounced	
		3	30	Severe	Died
		4	60	None	
		5	60	None	
		6	55	Pronounced	
14	8	1	60	None	
		2	60	None	
		3	60	None	
		4	55	None	
		5	60	None	
		6	60	None	
		7	60	None	
		8	55	Severe	Died

Cases described as "mild" shock showed accelerated respiration and in some cases restlessness. Cases designated as "pronounced" shock showed marked respiratory disturbances and lassitude and frequently nausea accompanied by vomiting; all of these cases recovered. Cases designated as "severe" shock were characterized by collapse and the symptoms in these cases are always alarming and the prognosis grave; 50 per cent of these cases succumbed.

vidual pigs were as follows: one pig, 25 per cent; four pigs, 30 per cent; two pigs, 35 per cent; two pigs, 45 per cent; five pigs, 55 per cent. It will be noted that severe shock occurred only in those pigs where the hemoglobin content of the blood was 55 per cent or below.

The foregoing summary and table show very clearly that there was a distinct correlation between the hemoglobin content of the blood and the degree of shock, in other words, the shock increased in degree or severity as the anemia became more pronounced. In the cases of severe shock, where collapse occurred, the shock resulted fatally in 50 per cent of the cases.

In the cases reported as "mild" shock, it is possible that the symptoms noted were due largely to excitement and fatigue resulting from catching and holding the pigs for vaccination, for it has been noted by Doyle that pigs suffering from anemia tire easily and "quickly become exhausted when made to exert themselves." Munce[9] states that symptoms of shock, which apparently resulted from the exertion of the pigs incident to catching and handling, have been reported to him by practicing veterinarians and it seems likely that in such cases the pigs were suffering from an unrecognized anemia.

In connection with the results just described, it should be stated that in all cases of severe shock with collapse, efforts were made to induce artificial respiration, with a view to saving as many of the pigs as possible, and it is probable that more cases would have resulted fatally had no restorative measures been taken.

Postmortem examination of the seven pigs which died from shock in this experiment revealed marked pallor of the subcutaneous and muscular tissues in every instance. There also was marked pallor of the internal organs, especially noticeable in the liver and kidney. In two cases, there was a considerable amount of fluid in the peritoneal cavity and a fibrinous exudate over the liver. In one case the spleen was much enlarged. The postmortem lesions as well as the hemoglobin tests in the fatal cases revealed a grave anemia.

Hemoglobin tests were made on the fourteen sows in this group. These tests showed a hemoglobin range of 85 to 95 per cent, with an average of 90 per cent, indicating that the sows were in good condition. It was thus apparent that the anemia in the pigs was not transmitted through the sows, but was a result of the conditions under which the pigs were held. As already stated, the sows and pigs in this group had been con-

TABLE II—*Summary of hemoglobin tests and shock reactions following vaccination in group 1. (See table I.)*

LITTER	PIGS	HEMOGLOBIN RANGE FOR LITTERS (%)	PIGS SHOWING SHOCK	DEGREE OF SHOCK			
				MILD	PRONOUNCED	SEVERE	
						RECOVERED	DIED
1	6	30-60	4	0	2	1	1
2	7	45-70	1	0	1	0	0
3	6	45-60	1	0	1	0	0
4	7	30-60	1	0	1	0	0
5	5	45-60	1	1	0	0	0
6	10	55-70	0	0	0	0	0
7	8	30-65	5	1	4	0	0
8	5	25-65	3	0	1	1	1
9	7	30.65	5	1	2	1	1
10	8	35-70	5	0	4	0	1
11	4	45-55	4	0	1	2	1
12	7	45-60	4	1	1	2	0
13	6	30-60	3	0	2	0	1
14	8	55-60	1	0	0	0	1
Totals	94		38	4	20	7	7
Average		30-70					

fined in small concrete pens or yards, where there was no access to soil, and no mineral matter had been fed. During the last three weeks of the experiment, the pigs had been confined in east and west individual pens, separated by board fences, with limited exposure to sunshine and limited amount of room for exercise. At the time these experiments were run, there was a good deal of cold, rainy weather and the sows and pigs remained in their houses a good part of the time. It is probable, therefore, that three factors were concerned in producing the anemia in this group of pigs, viz., lack of exercise, lack of sunshine and lack of mineral matter.

Group 2: Five sows with their litters (32 pigs) were placed in individual pens facing south. Each sow and litter occupied an individual pen. The pigs were of the same approximate age (3 weeks) and were placed in the individual pens within a day or two of the same date as those in group I and were fed in the same manner.

The pens were paved with concrete and were of the same size and type as those occupied by the pigs in group I, the only difference being that in this case the pens faced south instead of west and the pigs were thus afforded better exposure to sunlight. The pigs in this group were tested for hemoglobin and vaccinated at the same time as those in group 1, with the results shown in table III.

TABLE III—*Summary of hemoglobin tests and shock reactions following vaccination in group 2. These pigs had no access to soil and were fed no mineral matter. They had limited room for exercise, but good exposure to sunlight.*

Litter	Pigs	Hemoglobin Range for Litters (%)	Pigs Showing Shock	Degree of Shock		
				Mild	Pronounced	Severe
15	6	70-95	1	1	0	0
16	8	75-90	0	0	0	0
17	5	70-95	1	1	0	0
18	8	75-95	1	1	0	0
19	5	75-85	3	3	0	0
Totals	32		6	6	0	0
Average		70-95				

An analysis of the results shown in table III shows that in group 2, which consisted of 32 suckling pigs about 6 weeks of age, confined for the last 3 weeks in concrete pens affording a good exposure to sunlight, only six pigs (18.7 per cent) suffered from mild vaccination shock and in no case was the shock severe.

In this group of pigs, the hemoglobin content of the blood ranged from 70 to 95 per cent with an individual average of 81.5 per cent, which was a very much higher average than for group 1, where the hemoglobin ranged from 25 to 70 per cent, with a low individual average of only 52.5 per cent.

Hemoglobin tests, made on the five sows in this group, gave an average of 92 per cent, a slightly higher average than for the sows in group 1, which averaged 90 per cent hemoglobin.

In view of the fact that the only difference in the conditions under which groups 1 and 2 were kept consisted in a difference in the exposure of the pens which housed the two lots, the conclusion seems unavoidable that the higher hemoglobin average in group 2 was due largely to the increased amount of sunlight which these pigs received.

Owing to the fact that the pens faced south, however, and were thus exposed to sunshine, it is probable that these pigs were out in their yards most of the time and thus exercised more than the pigs in group 1, so that increased exercise also may have been a factor in building up a higher hemoglobin content in these pigs.

Group 3: Five sows with their litters (35 pigs) were placed in individual pens facing south, each sow and litter being given an individual pen. These pens were originally like those occupied

by group 2, but small dirt yards, about 7x10 feet, had been added at the back or north end of each pen.

The pigs in this group were tested for hemoglobin and vaccinated at the same time as those in groups 1 and 2, with the results shown in table IV.

TABLE IV—*Summary of hemoglobin tests and shock reactions following vaccination in group 3. These pigs had access to soil, ample room for exercise and good exposure to sunlight.*

Litter	Pigs	Hemoglobin Range for Litters (%)	Pigs Showing Shock	Degree of Shock		
				Mild	Pronounced	Severe
20	6	80-100	0	0	0	0
21	7	95-100	0	0	0	0
22	6	85-100	0	0	0	0
23	9	85-100	0	0	0	0
24	7	85-100	2	2	0	0
Totals	35		2	2	0	0
Average		80-100				

In this group of pigs, the hemoglobin content of the blood ranged from 80 to 100 per cent, with an average of nearly 95 per cent. Only two pigs in this group (5.7 per cent) showed symptoms of mild shock and in these cases the symptoms may have resulted from exertion incident to handling.

The higher hemoglobin average in these pigs, which was 13.2 per cent higher than for the last group, was undoubtedly due to the fact that these pigs not only had exposure to sunlight, but they also had nearly twice as much yard room for exercise and, in addition, what was even more important, they had access to soil in which to root and from which they could obtain essential blood-building mineral elements.

The following notes were made on the general condition of the three groups of pigs in the foregoing experiment up to weaning time:

On March 14, when eight weeks of age, a marked difference was noted in the general condition of group 1, as compared with groups 2 and 3. The pigs in group 1 were thin and flanky, as a rule, and were evidently growing tall but not filling out well and the hair on these pigs was not smooth. The pigs in groups 2 and 3, on the other hand, were much rounder and were evidently filling out better and the hair was smoother.

On April 15, when the pigs were weaned at about 12 weeks of age, the following notes were made: Group 1 showed a heavy death loss (23 per cent); the surviving pigs averaged well in

weight, but were uneven in size and the general condition and appearance of the hair was nothing like as good as those in the two other groups. Group 3 was well developed, even in size, with smooth hair; there were no runts in this group and the general condition of these pigs was superior to those in group 2, while the latter were much superior to those in group 1.

Concrete Lots Versus Dirt Lots for Young Pigs

It had been a mooted question at the ranch in past years as to whether it was better to carry young pigs till weaning time in concrete inclosures or yards, rather than in dirt yards. The owner of the ranch, in considering this matter from a purely sanitary point of view, reached the conclusion several years ago that concrete yards, which could be better cleaned, might prevent the development of infections up to weaning time. He had tried this plan with very indifferent success and had then abandoned the use of concrete brooder pasture pens but continued the use of individual concrete pens under conditions already noted.

As some of the old brooder pasture pens floored with concrete were still available, the following experiment was carried out with little pigs confined in two of these pens in comparison with pigs of the same size and age held in the regular brooder pasture dirt pens.

Each concrete brooder pasture pen or lot used in this experiment covered about 160 square yards. The houses or sheds were floored with wood and were open to the south, and the pigs were thus afforded ample sunshine and ample space for exercise, but had no contact with soil. The dirt brooder pasture pens were considerably larger, each covering about 500 square yards; the houses or sheds also were open to the south and the pigs in these lots thus were afforded ample sunshine, plenty of room for exercise, and access to mineral matter in the soil.

The experiment, which was begun on January 29, was carried out as follows:

Group 4: Twelve sows and their litters (80 pigs) were placed in two of the regular brooder pasture dirt pens, six sows and their litters being placed in each pen.

Group 5: Twelve sows and their litters (90 pigs) were placed in two of the concrete brooder pasture pens, six sows and their litters in each pen.

All of the pigs in these two groups were farrowed between January 6 and January 8 and were thus approximately three weeks of age when the experiment was begun. Prior to this

time, they had been confined in concrete farrowing-pens with their dams. In selecting the litters for this experiment, the endeavor was made to select as nearly uniform litters as possible, with regard to size and general condition. The sows and pigs were fed garbage and grain in the usual manner, as in the preceding experiment, and the growth and general condition of the pigs was observed from time to time.

Hemoglobin tests were made on 20 pigs in each group when the pigs were about nine weeks of age. These tests showed an individual average of 85.7 per cent hemoglobin for the pigs in the dirt lots and 78.2 per cent hemoglobin for the pigs in the concrete lots. At this time, there was quite a noticeable difference in the appearance and general condition of the two lots of pigs. The pigs in group 5, in the concrete yards, showed a considerable variation in size and about half of these pigs appeared to be underweight. The pigs in group 4, in the dirt yards, were quite uniform in size, had made a better gain in weight, and showed smoother hair than those in group 5. There was a difference of at least five pounds in the average estimated weight of the individual pigs in the two groups, in favor of the pigs held in the dirt yards.

The pigs in these two groups were given simultaneous treatment at the time the hemoglobin tests were made. No symptoms of shock were noted in the pigs in the dirt yards (group 5), which was not surprising, as the hemoglobin average for this group was 85.7 per cent and none of the pigs tested showed a hemoglobin content below 75 per cent. In the case of the pigs held in the concrete yards (group 4), several cases of mild shock were observed, but in no case was the shock severe; in this group the hemoglobin average was 78.2 per cent and in none of the pigs tested was the hemoglobin content below 55 per cent.

When the pigs in this experiment were weaned at about 13 weeks of age, the following notes were made: The pigs in the dirt yards (group 5) had smoother hair, were heavier, more uniform in size and weight, and appeared healthier than those in the concrete yards (group 4). A large proportion of the pigs in the concrete yards had "sway" backs and pigs showing this deformity are usually unsatisfactory to fatten.

This experiment showed very clearly, as did the preceding experiment, the marked benefit to be derived by placing young pigs in contact with soil from which they can derive the mineral matter essential to good growth and development.

SUMMARY

Experiments were conducted in which hemoglobin tests were made by the Tallquist scale on 201 suckling pigs six to nine weeks of age and the effects of serum-simultaneous treatment noted in these pigs. The pigs weighed from 20 to 40 pounds and received approximately 1 cc of clear, heated serum per pound. The virus dose was 2 cc. In this connection, attention is called to the fact again that all of the serum used in these experiments was clear serum, which had been heated in accordance with Bureau regulations governing the production of anti-hog cholera serum. The regulations governing the production of clear serum require a heating period of 30 minutes at 58 to 59° C. In every case, the serum was administered subcutaneously in the groin with a Shikles syringe and the virus was injected in the subcutaneous tissue over the ham. Many of the pigs in these experiments were anemic as a result of the conditions under which they were held. Symptoms of vaccination shock were noted in pigs which showed a pronounced anemia and a rather striking and close correlation was noted between the hemoglobin content of the blood and the degree of shock; in other words, the shock increased in severity as the anemia became more pronounced. From these observations, it is believed that anemia is an important factor in the causation of vaccination shock and affords the probable explanation of the occurrence of vaccination shock in veterinary practice, where shock has been observed on certain farms and not on other farms in pigs that have been treated with the same serum and virus on the same date.

Suckling pigs were held under varying conditions and an attempt was made to evaluate the importance of the three factors which play a part in the causation of anemia, viz., lack of sunshine, lack of exercise, and lack of contact with soil. The experiments recorded in this paper would seem to be in agreement with the results of other investigators who have studied the question of anemia in young pigs and would indicate that all three of the factors mentioned play a part in the causation of anemia and, of the three, lack of access to soil is the most important.

The fact that pigs were rendered anemic in these experiments through lack of sunshine, lack of exercise and lack of contact with soil, coupled with the fact that vaccination shock was noted in pigs which showed a marked anemia, would seem to offer an explanation of the fact that vaccination shock has been observed most frequently in veterinary practice in early spring pigs,

which had undoubtedly suffered from the three factors just mentioned and were therefore anemic.

It would seem that the apparently close correlation between anemia and vaccination shock in young pigs might prove to be a matter of some practical value to the veterinary practitioner in the vaccination of early spring pigs, which are apt to be anemic and therefore subject to vaccination shock. It would be a comparatively simple and easy matter, and one requiring very little time and involving but little expense, to make a number of hemoglobin tests, by means of the Tallquist scale, in a herd of spring pigs suspected of being anemic and thus determine beforehand whether severe and perhaps fatal shock might result from vaccination. If the pigs were found upon test to be quite anemic, it would be better to treat them first for the anemia before undertaking to immunize them with clear, heated serum. However, if hog cholera should be prevailing at the time and it should be imperative to immunize at once, the anemic pigs might be given blood serum. No cases of shock have followed the use of blood serum in experiments at Ames.

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Discussion

Dr. E. A. Cahill: I desire to compliment Dr. McBryde on his most interesting paper. Those of you who have been particularly interested in this problem perhaps appreciate more than others how valuable this additional information really is. During the past two years there has been developed considerable information explaining vaccination shock and demonstrating causative factors but, notwithstanding this additional information, practitioners continue to encounter some vaccination shock. The information contained in Dr. McBryde's report will, I am certain, be very valuable to practitioners throughout the country and particularly in the midwest.

I have just one question which I would like to ask Dr. McBryde. I would ask the question personally but I think that others will be interested in the same, and it may be helpful in promoting discussion.

Did you administer any whole-blood serum to the anemic pigs?

Dr. McBryde: No, Doctor, I am sorry to say that I did not. I would like to have had some whole-blood serum at the time the experiments were made.

Dr. Cahill: If a practitioner were working in a herd and he had

reason to believe that the pigs were afflicted with anemia and he must do something because of the immediate presence of cholera, would that practitioner be acting wisely to administer to those animals whole-blood serum instead of heated serum?

DR. McBRYDE: I think such procedure would be safe and that he would be less apt to get shock. I do not think that I have seen shock following the use of whole-blood serum. I would like to have had a chance to test parallel injections of whole-blood serum and heated serum on pigs showing marked anemia.

DR. R. L. WHITAKER: Has the method of injection any relation to the shock?

DR. McBRYDE: Yes, I think that shock is more apt to follow intraperitoneal injection. We found in our experiments at Ames that it is apt to be more severe following the intraperitoneal injection, and I understand that a good many veterinarians in Iowa have given up the intraperitoneal injection of serum for this reason.

Cellophane for the Veterinarian

That versatile transparent wrapping material, cellophane, may invade the field of veterinary surgery, if it has not done so already. It has been suggested that operative wounds might be covered with cellophane held in place by adhesive tape. This would leave the wound visible for inspection and also would exclude flies, dust and other foreign matter.

Do You Remember When

Dr. H. E. Kingman, of the Colorado Agricultural College, very proudly appeared on the campus at Fort Collins, behind the wheel of a new Franklin automobile? In the front seat with Dr. Kingman was Dr. Richard F. Bourne. In the back seat were Dean George H. Glover and Dr. I. E. Newsom.

IMPORTANCE OF DISEASE IN WILD-LIFE ADMINISTRATION*

By J. E. SHILLINGER, *Washington, D. C.*

In charge, Disease Investigations, Bureau of Biological Survey, U. S. Department of Agriculture

Increasing recognition of the importance of our wild life as a national asset has developed a concern, among able scientists in various fields, for the future of some of the valuable untamed animals and birds native to this country. Veterinarians are taking up the study of the ailments affecting many species, to coördinate their findings with the observations made by wild-life administrators, biologists and ecologists, with a view to preventing losses. Though unwise administration, the inroads by predators, and the effects of unfavorable food and weather conditions and of other obscure biotic factors may cause fluctuations in wild-life populations, it has become evident that disease is responsible for some of the more serious losses.

Most of the disease studies made thus far have been on animals maintained on fur and game farms or on preserves, where the stock is kept under controlled or semi-controlled conditions. A few outstanding investigations made of outbreaks of disease in the wild indicate that the cyclic disappearance of various species over wide areas is due to epizoötics that sweep over regions where conditions are favorable for the spread of infection. It is not the purpose here to give a detailed account of any one disease encountered in wild life, but to review briefly some of the outstanding causes of loss among the several groups of animals and birds.

For a number of years, the United States Bureau of Biological Survey has carried on investigations on diseases of fur animals in coöperation with the Department of Bacteriology of the University of Minnesota. Two distinct infectious diseases have been studied, both of which are sometimes spoken of as distemper, though neither is identical with the disease described by Laidlaw and Dunkin as canine distemper. One, which is caused by a filtrable virus and produces lesions in the central nervous system, is spoken of as fox encephalitis. (Fig. 1.) A fox affected with this disease may show lethargy, weakness, hyperexcitability, paralysis of certain groups of muscles, or convulsions—all nerv-

*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

ous affections, and their type depending on the particular location of the lesions. The same animal may exhibit two or more of these symptoms during the course of the malady, which is regularly very brief. The lesions may be macroscopic, but often they can be detected only with the aid of the microscope.

Ranch-raised foxes are subject to paratyphoid infections, and these, if unchecked, may kill a large proportion of the stock. Paratyphoid has a longer course than fox encephalitis, frequently running several weeks, during which there is a wasting of flesh and often a purulent discharge from the eyes and nose. On autopsy the most noticeable pathological change is a greatly enlarged, dark spleen. Experiments are being conducted for perfecting practical artificial immunity against these diseases.

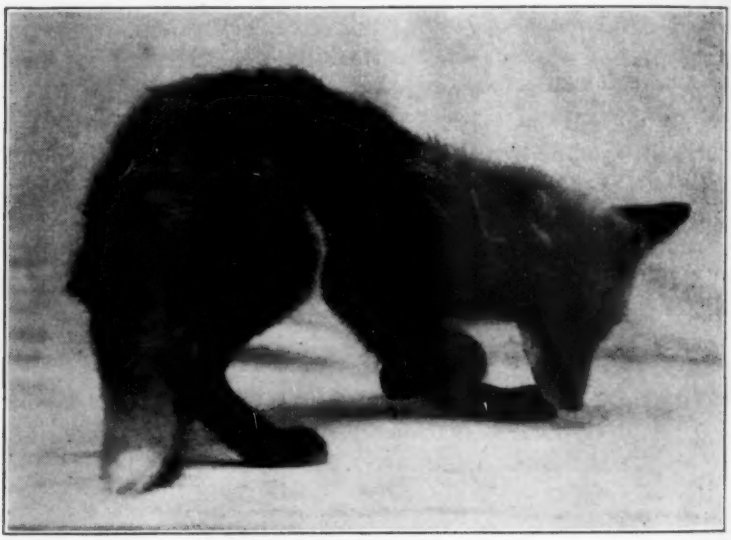

FIG. 1. Silver-fox pup affected with infectious encephalitis.

Another species that is raised for fur—the rabbit—is subject to a variety of diseases that are particularly disastrous among the young. A number of these have received rather extensive study. Coccidiosis and other forms of intestinal parasitism are satisfactorily controlled by means of well-known sanitary measures. Ear mange responds readily to simple preparations recommended for treating this condition in dogs and cats. Much remains to be done, however, on several infectious diseases that menace the rabbit industry. Myxomatosis is a rabbit disease caused by a filtrable virus introduced into this country during

the last few years. (Fig. 2.) It is highly infectious, when conditions are suitable for its transmission, and has a heavy mortality. In advanced cases there is a pronounced edema of the head and ears, often with the formation of excrescences of wart-like appearance on the margin of the ears and about the eyes and nose.

The best-known disease of rabbits in the wild is tularemia. The prominence of this animal disease is doubtless due to the fact that it is one that is transmissible and causes serious illness in man. Tularemia is generally associated with rabbits, and is sometimes spoken of as "rabbit fever," but it has been found as a natural infection in a great variety of other wild animals

FIG. 2. Myxomatosis in advanced stage.

and birds. On postmortem, the liver and spleen show small whitish spots, and in certain lymph-glands an enlarged hemorrhagic condition may be noted.

Though parasitism is probably not responsible for the spectacular and precipitous periodic declines in wild-life populations, its importance as a devitalizing factor and one causing pronounced local losses and the spread of infectious diseases, can not be overlooked. Deer, elk, moose and rabbits are frequently grossly infested with ticks. It is not uncommon to find as many as several thousand ticks on a single rabbit. Heavy tick infestations also are noted on moose and other large game animals at some seasons, and on some of our important upland game birds.

One of the most destructive diseases of big-game animals on preserves is necrotic stomatitis. Its appearance is most frequent where supplementary feeding is practiced to aid in carrying the animals over a winter of insufficient or unavailable natural forage. The causative organism, *Actinomyces necrophorus*, finds excellent places to grow in the accumulations of manure and waste products about the feeding grounds. Small abrasions in the mucous membrane lining the mouth, caused by harsh food substances, form a suitable portal of entry for the infection, and extensive destruction of tissue results. Often the mandibles are so grossly infected as to involve serious mechanical interference with nutrition.

FIG. 3. Silver-fox pup affected with rickets.

Nutritional diseases are not uncommon among wild animals kept under fully controlled conditions. In zoölogical gardens the animals frequently exhibit rachitic conditions, and fur-farmers have learned the importance of having a liberal supply of vitamin D in the rations fed. Heavily pigmented skin and hair appear to increase the susceptibility of animals to rickets, and it has been noted that in the Far North, where the sun's rays are less direct, or absent for long periods, the disease affects wild stock. (Fig. 3.)

In the northern part of this continent, a species of the warble fly, *Oedemagena tarandi*, is a serious plague among the reindeer.

Frequently there is a severe dermatitis on the legs where the adult insect deposits her eggs, and the fly larvae later burrow into the skin. The following spring the developed larvae often are found abundant just beneath the skin of the back and sides of the animals, and later the hides are perforated with holes wherever the larvae emerge. Screw-worm fly larvae (*Phormia terrae-novae*) also are a serious pest in that far-north country, and do extensive damage to the wounded animals they attrack. (Fig. 4.)

FIG. 4. Screw-worm infestation in reindeer.

Wild birds, especially migratory waterfowl are frequently subject to maladies that destroy them in great numbers. Recently several waterfowl disease centers have been subjected to detailed investigation. It has been found that the blood parasite, *Leucocytozoon anatis*, is responsible for extensive losses among young wild ducks in several parts of the country. The small blood-sucking black fly, *Simulium venustum*, is known to be a carrier of the infection, but it is not known that this insect is the only agency in the spread of the disease. If, however, this should prove to be the case, the disease will naturally be limited to its range. The blood of affected ducklings is thin, pale and watery and shows a great number of spindle-shaped protozoan parasites. On autopsy the spleen is found to be enlarged and dark.

A condition frequently involving extensive losses among waterfowl is the pollution of waters with oil and industrial waste. Oil, when spread on the surface of water in the form of a thin film, is not poisonous, but by adhering to the feathers of the birds it makes them matted and heavy. In this state the bird is unable to fly, and the matted condition of the feathers exposes its body to the cold water. Hence the death of waterfowl in cases of oil pollution is due to the results following the physical action of the oil and not to its toxicity.

On the other hand, several forms of chemical waste have been found poisonous to many birds feeding in waters where these products are discharged. In the vicinity of smelteries and factories, where waste chemicals from the industries are dumped

FIG. 5. Swans affected by chemicals in smeltery waste.

into adjacent bodies of water, it is difficult to avoid extensive losses of wild fowl that visit these water areas. (Fig. 5.)

Lead poisoning is common in wild ducks, geese and swans that eat shot picked up on marshes where shooting has been done extensively. It takes only a few shot in the gizzard to constitute a lethal dose of lead for these birds. The soft metal is rapidly ground up by the action of grit in this organ, and since the finely divided lead becomes soluble in the presence of the digestive fluids, absorption takes place rapidly. A greenish diarrhea, accompanied by weakness of certain muscles, especially in the carpal joints, is the predominating symptom. The results of paralysis of the gizzard muscles are frequently evident on

necropsy. Inability of the gizzard of an affected bird to handle the ingested foods results in a distention of both proventriculus and esophagus. Aside from the actual deaths caused by such lead poisoning, it has been reported that sublethal doses of lead may cause sterility. Tests are being conducted to learn what effect, if any, small doses of lead may have on reproduction in these birds.

One of the most important studies on diseases of wild life made by the Bureau of Biological Survey in recent years is that of botulism in waterfowl and shorebirds. This condition was formerly referred to as alkali poisoning, because it has made its appearance most commonly in shallow alkaline lakes in the

FIG. 6. Shore of a western lake showing great numbers of sick and dead ducks as a result of botulism.

western part of this country. During the latter part of the summer, the action of the warm sun, on shallow waters having an alkaline reaction and containing an abundance of decaying vegetable and animal matter, furnishes suitable conditions for the growth of *Clostridium botulinum*, type C. Botulinus toxin has been demonstrated in a variety of organic substances found in these lakes upon which the waterfowl and shorebirds ordinarily thrive. (Fig. 6.)

Upland game birds also are subject to a variety of diseases, some of which have been known as common ailments in domestic fowls. Blackhead, coccidiosis, fowl-pox and tuberculosis act much the same in quail and grouse as they do in domestic chickens and turkeys. Conditions not common to farm stock, however, are found in upland game birds. In quail a malady spoken of as "quail disease," perhaps more properly designated as ulcerative enteritis, takes a huge toll on many game farms. The outstanding lesions in affected individuals are ulcerations in the mucosa of the intestine. These are usually noted in the small intestine and may extend its entire length. Sometimes the ceca also may be seriously affected. In the early stages of an outbreak, however, few pathological conditions may be found in the intestine, the first birds frequently dying before the develop-

FIG. 7. Cat nursing an orphaned litter of silver-fox pups.

ment of extensive ulcerations. The causative organism has not yet been identified, and no satisfactory treatment is known. Since the infection appears to be transmissible in the feces of affected quail, an outbreak may be checked by isolation of the affected birds and the exercise of strict sanitation.

In wild-life administration it is not feasible, with our present knowledge of the diseases of the various species, to control losses completely. On fur and game farms, where the stock is kept under penned conditions, significant headway has been made in eliminating infectious, nutritional and parasitic diseases. On large game preserves, where the animals and birds are under a semi-controlled condition, certain methods may be adopted that will so improve the environment as to afford fairly efficient pro-

tection against some of their diseases. These practices may even be found applicable to conditions in the wild.

In some cases much may be done by administrators in the rescue of helpless individuals. An example is found in the wholesale rescue work that is being done among wild ducks affected with botulism on western lakes. Most of the birds are found in a weakened state and unable to fly, but they usually recover when placed in a healthful environment, away from sources of toxin. Young orphaned animals also are frequently saved by the use of foster mothers. The cow will perform the function for various young wild ruminants, such as buffalo and deer. Most fur-farmers have learned that a docile domestic cat can be utilized satisfactorily for raising orphaned silver-fox pups. (Fig. 7.)

With the increased interest and activity of administrators and scientists in studying wild-life management, including disease control, it is anticipated that an increased efficiency in protecting the various species in the wild will be developed. Veterinarians in regular practice, as well as those at state colleges and experiment stations, are in strategic positions to coöperate with conservation officials, game-farmers and fur-farmers in working out improved measures for the prevention and control of diseases in wild life.

Putting Vitamin D in Milk

It has been conclusively demonstrated that milk given by cows whose product ordinarily shows but from 4 to 8 Vitamin "D" effectiveness may have this effectiveness multiplied, making it from 16 to 30 times as great, through having their rations supplemented with the prescribed amount of irradiated dry yeast.

Certified Milk.

BUREAU TRANSFERS

Dr. M. W. McGuire (K. C. V. C. '08), from Joplin, Mo., to Terre Haute, Ind., in charge of meat inspection.

Dr. R. J. Spain (Gr. Rap. '18), from Reno, Nev., to Sacramento, Calif., on tuberculosis eradication.

Dr. Roy Avant (A. P. I. '16), from Atlanta, Ga., to Tuscaloosa, Ala., on meat inspection.

Dr. W. C. Herrold (O. S. U. '07), from Cincinnati, Ohio, to Omaha, Neb., in charge of meat inspection.

Dr. W. Dean Wright (Chi. '03), from Ogden, Utah, to Tacoma, Wash., on meat inspection.

Dr. Albert R. Miller (Iowa '24), from Jersey City, N. J., to South Kortwright, N. Y., on meat inspection.

A CONTINUATION OF THE STUDY OF THE ETIOLOGY OF INFECTIOUS DIARRHEA (WINTER SCOURS) IN CATTLE*

By F. S. JONES, RALPH B. LITTLE *and* MARION ORCUTT

Department of Animal and Plant Pathology,
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Princeton, N. J.

In a previous paper[1] we described a communicable disease of cattle in which diarrhea was the principal symptom. The disease occurred in epizoötic form in certain herds in the late autumn and winter, and usually attacked all the animals in a given group or all those in the herd, except the younger calves. The cause of the disease proved difficult to discover, but late in the season, when no more material was available, the bacteriological findings indicated that vibrios were responsible. In order to confirm, if possible, the results obtained from our examinations of materials from the last three outbreaks in 1929, the disease was studied anew.

In our first paper we called attention to the fact that when calves were fed feces from spontaneous cases, severe disease never resulted, but a well-recognized chain of events followed. As a rule there was a slight rise of temperature (about 1° C.) during the first 24 hours; the stools then became soft, large quantities of mucus appeared in the feces and, if the animal were slaughtered, a mild but well-defined involvement of the small intestine was found, and vibrios might be cultivated from the inflamed mucosa.

THE OCCURRENCE OF THE DISEASE DURING THE AUTUMN OF 1929 AND THE WINTER OF 1930

The first outbreak of diarrhea occurred in November, among a group of newly purchased cows in a large dairy herd. The disease showed no tendency to spread to the main herd. Certain cows scoured severely for several days, while adjacent ones remained unaffected. Bloody stools containing mucus were the rule and, in certain cases, the animals passed what were described as casts of intestine.

In another instance, two newly purchased cows that had been introduced into a small herd developed diarrhea within 12 hours.

*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

The disease spread to six others which scoured severely. Later, younger cows were introduced into the barn and were attacked promptly, and from these the disease spread to those that had not been affected as a result of the first exposure. In all, 37 of 43 cows were afflicted.

In a third outbreak the disease spread more slowly from cow to cow until every adult contracted it. Severe diarrhea appeared in a fourth herd but was confined to relatively few animals.

FIG. 1. Fragments of mucus obtained from the feces of a spontaneous case of diarrhea. (Actual size).

In all instances the symptoms were comparable; the feces were liquid and copious and contained in many cases, considerable quantities of blood, mucus (fig. 1) and blood-stained mucus. Microscopic examination (figs. 2 and 4) of this material revealed vibrios in enormous numbers. Occasionally vibrios were cultivated from this material (fig. 3). The disease as a rule, was afebrile and usually accompanied by inappetence, depression, and a greatly decreased milk yield. There was no mortality.

EXPERIMENTAL

Although vibrios could be demonstrated in the feces of spontaneous cases, it was with difficulty that they could be cultivated. In most instances it was necessary to reproduce the disease by feeding calves the feces obtained from the spontaneous disease and culturing the small intestine at the proper time. Many of the calves used in the experiments were removed from the dams after a few days and reared at the Institute. A few, when two to three months old, were purchased from small herds nearby. All were free from spontaneous intestinal disorders. The results

TABLE I—*The effect of feeding feces from spontaneous cases of diarrhea to healthy calves.*

ANIMAL	AGE	CLINICAL AND PATHOLOGICAL FINDINGS	BACTERIOLOGICAL FINDINGS
1692	8 mos.	Rise of temperature within 24 hrs. Inappetence. Feces began to soften after 48 hrs., diarrhea 3rd day, mucous casts containing vibrios in the feces. Slaughtered 3rd day. Gastro-enteritis	Vibrios in pure culture from the jejunum and ileum
1694	8 mos.	Irregular temperature during ensuing 4 days. Feces soft and loaded with mucous casts containing large numbers of vibrios. Slaughtered 4th day	Vibrios cultivated from the mucus in feces but not from materials obtained at autopsy
1702	5 mos.	Began to pass mucous casts containing vibrios after 48 hrs. Continued to do so until the 4th day, when the mucus contained leucocytes and epithelial cells. Involvement of the jejunum at autopsy on the 4th day	Vibrios mixed with other bacteria in cultures from the mucosa of the jejunum
1708	5 mos.	Rise in temperature during 1st day; feces soft at that time, containing mucous casts 8 cm. long. On 2nd day feces softer and containing casts a meter or more in length (fig. 5), composed of thin tenacious mucus with enormous numbers of vibrios embedded (fig. 6). Autopsy on 3rd day showed involvement of small intestine	Vibrios (fig. 7) cultivated from the mucous casts. Media inoculated with inflamed intestinal mucosa at autopsy remained sterile
1736	Unbred cow 5 yrs. old	No reaction	Vibrios not found
1757	24 days	No clinical reaction. Slaughtered after 48 hrs. Severe involvement of the jejunum	Vibrios in pure culture from the jejunum (fig. 8)

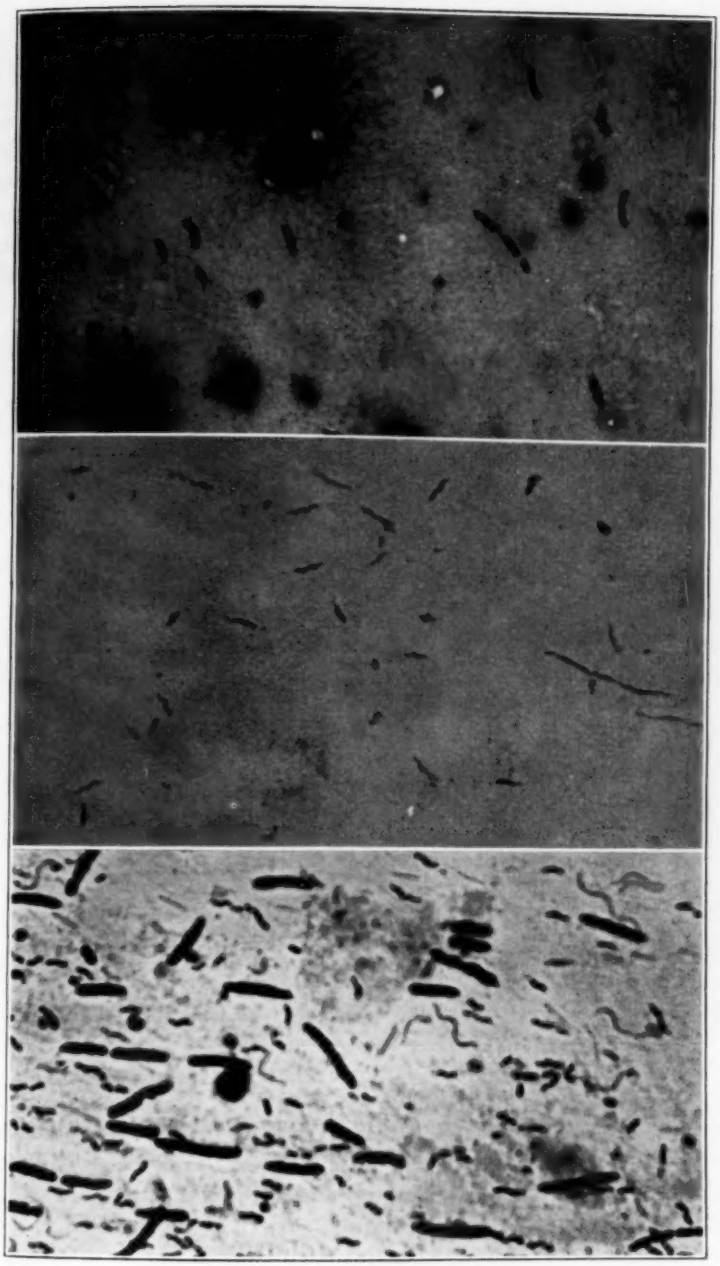

FIG. 2 (above). A portion of the same material, mounted and stained with Giemsa ; note the curved, irregularly convoluted vibrios (x 1660).
FIG. 3 (center). Original blood-agar culture (Br. 4) obtained from the fecal mucus shown in fig. 2; Giemsa stain (x 1660).
FIG. 4 (below). Fragment of mucus from another outbreak, mounted and stained with Giemsa. Well-stained vibrios are visible (x 1660).

of feeding feces from naturally occurring cases of diarrhea are recorded in table I.

Of the six animals fed material from the spontaneous cases, five reacted in a significant manner and one failed to react at all. The clinical manifestations were slight but the appearance of mucus, usually as casts (fig. 5) containing vibrios in large numbers, was significant. In addition, vibrios were cultivated either from the cast material or from the involved small intestine in all

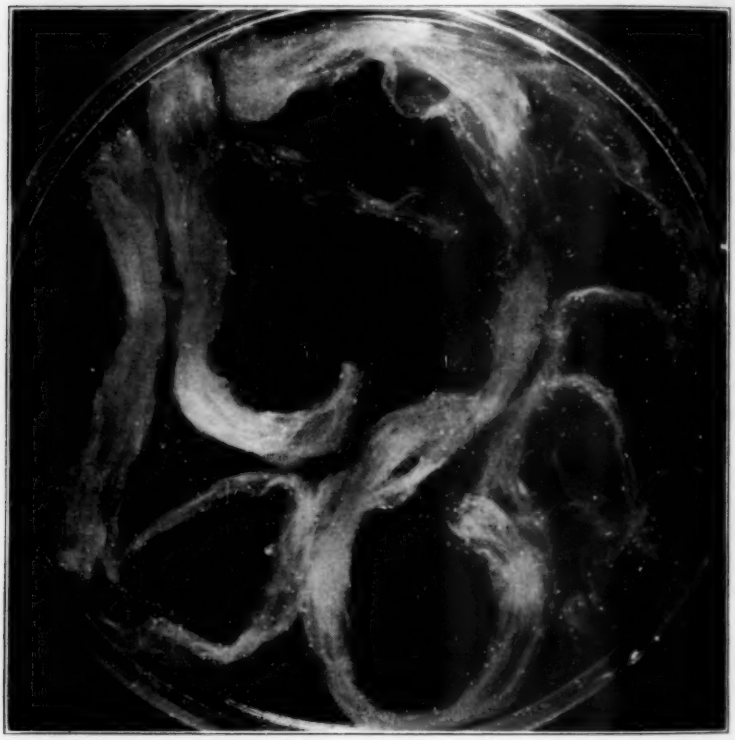

FIG. 5. Mucous casts found in the feces of calf 1708. (Actual size).

five instances. Cow 1736, which failed to react, was fed feces from the last case to occur in a small herd. The case was convalescent when the material was obtained and this fact might explain the negative result, although we had no assurance that cow 1736 had not passed through the disease in earlier life.

In addition to the four pure cultures of vibrios obtained by inducing the disease in calves, it was possible to cultivate such organisms (figs. 2, 3 and 4) directly from the mucus found in

the feces of a few spontaneous infections. Another successful cultivation was made from the upper ileum of a cow slaughtered at the height of the disease. In all, six strains of vibrios were available. Practically all strains grew poorly at first and growth

TABLE II—*The effect of feeding pure cultures of vibrios.*

CALF	AGE	FED CONDENSATION FLUID, BLOOD-AGAR SLANT CULTURES	RESULTS
1695	10 mos.	Vibrios 1692, 1694, B_4, 1708	Rise of temperature, 24 hrs. Large quantities of mucus in feces on 2nd day. Vibrios present in large numbers, feces soft, black on 4th day. Autopsy, 5th day. Well-defined enteritis. Vibrios in mixed culture from the feces but not from material at autopsy
1744	2½ mos.	Vibrios 1692, B_4, 1708	Rise of temperature, 24 hrs. Feces soft and brownish black in color. Large quantities of mucus evacuated, patchy congestion of jejunum at autopsy on 4th day. Vibrios not obtained
1754	3 mos.	Vibrios 1692, 1694, 1708	Rise of temperature, 24 hrs. Feces soft, dark brown, fetid, semi-solid; mucous casts after 48 hrs. Autopsy, 3rd day. Well-defined inflammation of the jejunum. Vibrios in mixed culture from jejunum and liver
1765	3 mos.	Vibrios 1692, 1757, 1763	Rise of temperature, 24 hrs. Mucous casts in soft and semi-solid feces; vibrios present in the mucus. Autopsy, 3rd day. Well-defined enteritis. Mucosa of jejunum overlaid with tenacious mucus. Vibrios in pure culture from jejunum
1771	3 mos.	Vibrio 1757	Feces soft and contained mucous casts on 4th day; 5th and 6th days, feces soft, chocolate-colored and contained mucus in large quantities. Vibrios in large numbers in the casts. Well-defined inflammation of duodenum, jejunum. Vibrios in pure cultures from upper jejunum, lower jejunum and upper ileum

was confined to the blood-condensation-fluid mixture at the bottom of the sealed agar slants. After several transfers, the number of vibrios increased in the cultures. Of the six strains, five were either S-shaped or undulated and the sixth was straight. All presented the characteristic motility and all tended to become beaded or fragment after active growth.

Immunologically the group, as judged by agglutination, was composed of distinct entities, thus differing from the vibrios found in the previous investigation. The actual relationship of these vibrios to the disease is brought out in table II. It will be noted that in most cases the cultures were combined and fed to calves.

Of the five calves fed cultures, all manifested recognizable symptoms; a slight rise of temperature usually occurred; the feces became softer, darker in color and contained large quantities of mucus. In four instances vibrios were recognized in the mucus and twice they were found in the cultures made from this material. In two instances we succeeded in recovering the culture from the inflamed intestine at autopsy. Calf 1744 reacted characteristically but the vibrios were not demonstrated.

In another experiment a cow, when fed the growth from two blood-agar cultures of a single vibrio (strain 71), developed mild diarrhea characterized by liquid feces which contained excessive quantities of mucus in which vibrios were demonstrated.

It seemed clear that the seasonal diarrhea which we have described was caused by vibrios which might frequently be demonstrated in the mucus appearing in the feces, but could rarely be cultivated from such material. In addition, vibrios had been found in the intestinal tract of a cow early in the spontaneous disease. When the condition was artificially induced, as when feces from acute cases were fed to calves, the organism produced definite disturbances and appeared in the mucous casts of the intestines and might, under the best conditions, be cultivated from inflamed portions of the small intestine. Pure cultures of vibrios, when administered to calves by mouth, produced the same effect.

DISCUSSION

The diarrhea which we have observed in this vicinity has a seasonal distribution. As a rule, milch cows suffer more severely than the younger animals. From the experiments reported it must be recognized that the disease is a communicable one caused by vibrios. The vibrios, while possessing certain characters in common, cannot be regarded as a compact immunological entity.

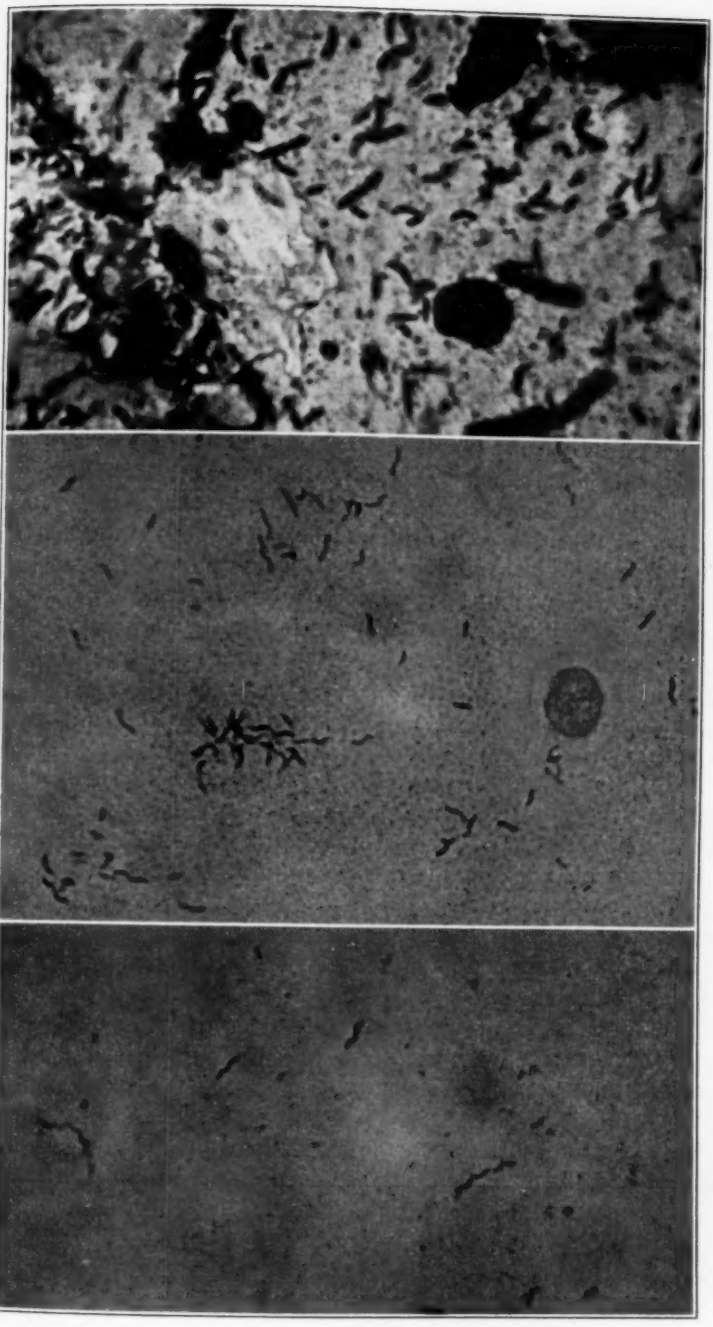

FIG. 6 (above). A fragment of preceding, mounted and stained with dilute carbolfuchsin for 8 hours. Note the heavily stained curved and comma forms (x 1660).

FIG. 7 (center). Culture, after thirty transfers, obtained from the material shown in fig. 6 (x 1660).

FIG. 8 (below). Vibrios, original culture, calf 1757, Giemsa stain (x 1660).

From the experimental infections and clinical observations, as well as a few autopsies of cows spontaneously affected, the disease process may be considered thus: Vibrios when ingested readily pass to the small intestine where, for a short period, they multiply rapidly. During this phase there is a slight febrile reaction but no diarrhea. The vibrios excite copious mucus secretion, so that the mucosa becomes overlain with mucus, vascular changes become pronounced, the mucosa becomes more permeable, permitting the passage of fluids, and the mucus in the form of casts is thus freed and diarrhea is apt to result. If, as we have demonstrated in certain cases, the whole cast rich in vibrios passes out in the feces, the individual promptly recovers, although diarrhea may persist for a day or two. In more severe cases it is possible to argue that the mucosa is more severely involved and the superficial layer sloughs with the attached mucus so that the capillaries discharge directly into the intestinal lumen, thus accounting for the appearance of blood in the feces and the peculiar chocolate or black appearance of the excrement. It is not impossible that vibrios similar in certain respects to those which we have described may play some part in clinically similar diseases of unknown etiology in man, especially as the usual methods employed in the investigation of such diseases would not lead to their detection.

With regard to the epizoölogy of infectious diarrhea, little is known except that the disease appears in the colder months and may be propagated by the introduction of cows recently infected. In certain instances it has been difficult to trace its origin. The vibrios possess relatively little pathogenicity and are apparently rapidly eliminated from the small intestine. The existing evidence suggests that the organism is not a primary parasite of the cow's small intestine. It is possible that, after the vibrios are eliminated from the small intestine, they may survive in small numbers in the large bowel and the disease thus is propagated from season to season. It may be true also that subclinical infections occur more frequently than the well-recognized type and a mild disease may be carried on until a sufficient number of relatively susceptible cows become available, when the disease may appear in epizoötic form.

SUMMARY

The continuation of the study of the etiology of seasonal diarrhea in cattle indicates that the disease is an infectious one. Vibrios of general morphological and cultural similarity were

observed in mucous casts found in the feces of spontaneous cases. In one instance they were cultivated from this material and in another they were cultivated from the inflamed intestine of a spontaneous infection slaughtered early in the disease. In addition, strains were cultivated from mucous casts in the feces and inflamed small intestine of calves in which the disease had been induced by feeding fecal suspensions from spontaneous cases. Pure cultures, when fed to calves, gave rise to well-defined intestinal disturbances and, in certain instances, the vibrios could be recovered from the inflamed intestinal tract and frequently demonstrated in the mucus found in the feces.

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[1]Jones, F. S., and Little, R. B.: Jour. Exp. Med., liii (1931), p. 835.

Control of Horse Parasites Becoming Popular

Treatment for the elimination of bots and other internal parasites of 21 horses of the military stables of the University of Illinois resulted in an average monthly gain of 46 pounds per horse the first two months following treatment, university officials recently reported. One horse, which was to have been killed because of its unthrifty condition, gained 255 pounds in weight in the 6 months following treatment, and proved to be one of the best mounts in the stables.

The benefits of parasite control extended to 20,857 farmers in Illinois. Drs. Robert Graham, E. E. Slatter and S. E. Park, of the University of Illinois, directed the campaign. Preliminary reports indicate that 90 per cent of the farmers who coöperated were pleased with the results. The treatment appeared to improve the condition and, incidentally, the capacity of the animals for work.

Iowa inaugurated the first state-wide campaign for the control of horse bots and other internal parasites of horses. The movement spread to Illinois and other states. Usually the work is organized in a township or county by an extension worker in coöperation with farmers and horse-owners of the area selected for the campaign. This arrangement cuts down the cost of the treatment, which is administered by veterinarians trained for this work. The control methods used in these campaigns have been developed and standardized by the U. S. Department of Agriculture.

SOME CASES OF DIABETES IN DOGS*

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The term diabetes is derived from a Greek word meaning a syphon, or, literally translated, "flowing or running through." Hence its application to the increased excretion of urine. There are, as is well known, two forms—diabetes insipidus and diabetes mellitus. The two forms were recognized and differentiated early. The first was characterized by a large amount of urine of a low specific gravity and insipid taste; the latter by a copious amount of urine with a honey-like, sweetish taste. This paper will deal with only the latter variety and will be limited almost entirely to a discussion of some reports of cases in which sugar was found in the urine.

Diabetes mellitus is defined by Osler as a disease of metabolism, particularly of the carbohydrates, in which the normal utilization of the carbohydrates is impaired, with an increase of sugar in the blood and consequent glycosuria.

There is little in veterinary literature upon the subject except that the disease is usually briefly described in veterinary texts. Law's "Veterinary Medicine" (Vol. II) devotes almost exactly two pages to the subject. Among other things, he states that the disease usually appears in old, pampered, fat dogs, especially affected with dyspnea or asthma, with dysuria and lameness, loss of weight, corneal ulcers, and cataract.

Müller and Glass also give two pages to the subject. They state that the disease is not exactly rare in dogs. Among the symptoms mentioned are enormous appetite but emaciation, easy fatigue, thirst, increased urination, cataract in one or both eyes, ulcerated keratitis, falling of hair, vomiting, diarrhea, ulceration of the skin, and bronchitis.

Jacob gives the disease only a page and a half, and states that the disease is not common. Among its complications, he mentions cataract, ulcers of the cornea, bronchial catarrh, gastric and intestinal catarrh, and eczema with alopecia.

Hoare describes the disease in all species in a few pages. He states that all authors regard the disease as being rare, the majority being recorded in the dog. He makes the observation that undoubtedly more cases would be diagnosed if more urine examinations were made by clinicians. He gives the symptoms about the same as the other writers, but stresses eczematous

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conditions of the skin of dogs, and in some cases catarrh of the external genito-urinary organs.

We are inclined to agree with Hoare, "that more cases would be seen if clinicians made more urine examinations," but from our considerable number of urinary examinations, and comparatively few cases of diabetes, we believe that the disease is not common.

We do not make as many urine examinations as we should, but do resort to them in cases in which a diagnosis cannot be made by a physical examination, or in an endeavor to learn why some apparently simple cases do not respond to treatment. In this way, we have occasionally encountered diabetes, and often when we least suspected it.

We did not make blood sugar tests, in the cases to be described, for various reasons, and consequently it may be better to think of them as cases having sugar in the urine, rather than as diabetes mellitus.

For several years, I have noticed what appeared to be some connection between the presence of sugar in the urine and some skin and eye lesions, especially alopecia of the former, and ulcerative keratitis in the latter. Whether there is any true connection, or whether our cases have simply been coincidences, I am unable to say. At any rate, it seems best to record some of our experiences. Most of these cases have resulted from our effort to find out what was responsible for certain obscure conditions, and often common conditions. With very few exceptions, the histories did not point to diabetes, nor did the condition of the animal suggest it. Its diagnosis might be called accidental. In other words, while we think of all the things that can happen in diabetes, we do not often connect a case brought in on account of a cough, or an ulcer of the cornea, eczema, or alopecia, as being diabetic until the condition does not respond to the usual treatment, and we begin to look around to see if we can find why.

Case 1

The first case to be described is the only one in which large amounts of sugar were found, the only one in which the physical condition of the animal was greatly affected, and the only one for which our records cover any length of time. The history here, even, did not point to the disease.

The subject was a black and white, female, 11-year-old fox terrier. Previous history was good. Several months previously, a mammary tumor had been removed under morphin and local anesthesia. Healing had been prompt.

On July 23, 1922, she was brought to us on account of a cough and loss of condition. With the exception of a slightly hurried respiration and the cough, she appeared perfectly normal. The cough had persisted for several weeks. Auscultation gave an increased respiratory murmur, percussion was negative, and temperature normal. The cough could be induced easily by a little exercise, or by pressure upon the trachea or larynx. A diagnosis of chronic bronchitis was made, and the animal put on creosote carbonate and calcidin in tablet form, and a mixture of ammonium chlorid in syrup of wild cherry. She appeared to improve on this treatment, and more than held her own during the summer and fall, with the exception of some more or less acute attacks, until the following February, when she had a few bad spells which steaming seemed to relieve. In March, her breathing was very bad, and she tired easily. She had a good appetite, but had a great thirst and voided an increased amount of urine.

We could not see this animal on account of her being so far away, and had to do everything by correspondence. At this time, I suggested a change of diet, and prescribed digestives. She picked up on this treatment, and, on April 12, was reported feeling better and stronger, but still easily exhausted. A neighboring veterinarian examined her and said the heart was off. On April 19, the animal was reported better. At this time, I suggested a urine examination. The first sample was examined April 25. It contained 38.6 gms. of sugar per 1,000 cc. Her diet was then changed to eliminate most of the starches. She appeared to gain, but on May 11, the urine showed 43.5 gms. per 1,000 cc, plus a positive test for acetone and diacetic acid. Her diet consisted of gluten bread, eggs, meat, asparagus and spinach. Her owner and a local physician, who was helping to keep track of her, thought she was much improved over two months before. She kept in good condition on this diet, and the sugar dropped to 33 gms. per 1,000 cc.

During the latter part of July, she was not so well, and it was decided to give insulin, which had just been put on the market. On August 6, insulin was started with one-unit doses, morning and night. About this time, her eyes failed. On August 10, the insulin was increased to two units, twice a day. On August 13, the insulin was increased to three units. On August 16, the sugar was 33 gms. per 1,000 cc. Acetone and diacetic acid were diminished. September 25, there were 70.5 gms. per 1,000 cc, a trace of acetone, but no diacetic acid. At this time she was doing well in every way but her sight. The insulin was in-

creased to 7.5 units at a dose, the bad breath disappeared, her coat was soft, legs strong and filled out, nose cold and moist, and she was acting in a nearly normal way. Cataract was diagnosed by the physician.

On October 8, a specimen of urine showed 70.5 gms. of sugar. On October 31, the dog had a bad upset. The insulin was increased to 17 units before each meal. On November 3, the urine showed 82.4 gms. of sugar, more acetone and more diacetic acid than before. November 6, 75.4 gms. of sugar. The diet consisted of 3 ounces of meat, an egg, lettuce and gluten gems fifteen minutes after the injection of the insulin. Her weight was 22 pounds—about two pounds below normal.

She was given up as hopeless at this time, and destroyed.

Case 2

On March 20, 1930, a three-year-old female wire-haired fox terrier was brought in suffering from an irritation of the right eye. The owner stated that the eye had been irritable, and that the pupil had been dilated for two weeks.

The cornea and lens were clear, the pupil widely dilated, and there were shreds of pus or tissue extending down from the posterior side of the iris across the pupil. There was an area of inflammation upon the upper side of the iris.

A diagnosis of iritis was made. The animal was put on small doses of potassium iodid, and a 1 per cent arecolin solution locally to constrict the pupil, and to protect the retina from the light.

We did not see this animal again until July 3, when she was brought in on account of a digestive upset. The eye appeared about the same as at the previous examination. The tissue mentioned above was evidently detached retina. A urine examination was suggested. This was made on July 5, at which time 2.9 gms. of sugar per 1,000 cc was found. Her diet was changed to gluten bread, meat, eggs, etc. On August 30, the eye was enlarged and soft, evidently a hydrophthalmus. Arecolin solution was used again to constrict the pupil and lower pressure.

By September 20, the case was more acute, the eye large and hard, and the animal apparently in considerable distress. On October 25, the eye was large and tense. The lens had been displaced and could be seen easily upon the floor of the anterior chamber. The eye was clear but large. For a considerable time, there had been quite regular periods, averaging almost exactly every three weeks, in which the eye appeared to pain the animal.

The owner expressed the opinion that the arecolin solution gave the animal relief, and hastened the end of the acute attack.

The night of December 1, the cornea of the left eye became inflamed. Upon examination, the cornea was blue, the pupil was widely dilated, and did not react to light. The cornea was too cloudy for a thorough examination. Arecolin solution was dispensed to constrict the pupil, and the owner was advised to use boric acid packs several times daily, and to keep the animal in the dark. The right eye was in about the same condition in which it had been for several months. The superficial keratitis in the left eye cleared up satisfactorily.

On February 1, there was an ulcer 3/16 inch in diameter on the left cornea. This was treated several times daily with warm boric acid packs, followed by instillation of a 1 to 5,000 metaphen solution. The ulcer also was outlined two or three times a week with fluorescein solution.

By February 8, the ulcer had improved. There was less staining, and its edges were becoming rounded. By February 15, the external layers of the cornea had given way, allowing Descemet's membrane to protrude, up to a level with the external surface of the cornea. The edges of the ulcer appeared to be rounding, but blood-vessels had grown over the entire surface of the cornea. The eye was increasing in size, and the patient was in some distress. The eye was treated with a 1 per cent solution of atropin and a 1 to 5,000 solution of metaphen.

The animal was destroyed somewhat later, as it was entirely blind.

Comment on Cases 1 and 2

The first of these cases showed much interference with nutrition, but not until fairly late in the disease. No doubt an earlier examination of the urine would have helped. It proved to be one of those cases which it is easy to look back at, and think what might have been done, but never will be done until someone makes a business of the routine examination of the urine. Case 2 was always in good condition, with the exception of the eye trouble. Her diet was carefully looked after. The urine examination was made because the eye lesion did not respond to treatment, and we believe we found why. More urine examinations should have been made.

Case 3

A six-year-old male Manchester terrier. The owner stated that the dog had been blind in one eye for nearly two years, and in the other for four months. Examination showed the dog in

good condition but blind. There were very opaque cataracts in both eyes, and the pupils were widely dilated. There was a loss of hair over the face, and on the inside and back of the hind legs. The skin in these areas was pliable and soft with no symptoms of inflammation. Scrapings were negative. Urine examination showed 5 gms. of sugar per 1,000 cc of urine.

Case 4

A six-year-old female Boston terrier. She had been ailing for several months. The owner reported that the dog had been drinking and urinating too much. The abdomen had been distended two months previously.

Examination showed the dog to be in fair condition. There was nothing abnormal except a loss of hair or baldness, especially around the thighs and upon the body. The skin was smooth, soft and pliable. Scrapings were negative. Urine examination showed 1.1 gm. of sugar.

Case 5

A female chow about four years old. The dog seemed to be in good health with the exception of loss of hair on the back just anterior to the tail, and upon the back sides of the hind legs. Scrapings were negative. The skin in the areas above described was soft and just seemed to be bald. We made a diagnosis of chronic eczema, and treated the dog for this condition, but with no improvement. Finally, the owner gave a history of the animal being very thirsty and urinating excessively. Urine examination showed a considerable amount of sugar.

Case 6

A middle-aged Airedale terrier in fine condition, with the exception of absolute baldness all over his saddle. This animal was in good condition. Urine examination showed sugar.

Case 7

A one-year-old Irish setter. There was no history in this case except a loss of weight, on account of which the animal had been wormed several times.

Examination showed that the animal was very thin, but in good lively spirits. Nothing abnormal could be found. Urine examination revealed a considerable amount of sugar.

Since the animal belonged to a physician, he was advised to put the dog on the same kind of a diet as he would a person. The dog gained rapidly on this change of diet, but was stolen after a few months.

Comment on Cases 3 to 7

Cases 3 to 7 are not complete enough to show more than that a urine examination may be an important aid to diagnosis and prognosis. Whether there was any connection between the sugar in the urine and the condition of the animals cannot be definitely proven, but there is, at least, a suggestion.

Summary

This paper is not sufficiently complete to warrant any conclusions. It is likely that there is some connection between the presence of sugar in the urine and certain cases of alopecia and eye lesions. This series of cases is presented simply for what they are worth.

In closing, may I urge upon you all the need for more case reports. A larger literature upon the diseases of small animals is needed, and a record of our observations is necessary. You practitioners are the ones to supply the information.

Discussion

Dr. C. F. Schlotthauer: I have seen many cases of artificially produced diabetes in the dog and the skin condition and loss of weight are very typical. It seems that the skin loses its resistance to infection and there may be a loss of hair and emaciation.

Dr. J. W. Patton: I was very much impressed with the case of a dog that apparently was diabetic. I mentioned it to a physiologist. He stated, "If you have a diabetic dog, you will not have to teach in a veterinary school. You will have them coming from all over Europe to see a genuine diabetic dog." I think we ought to be very careful in speaking of a diabetic dog. We are producing the condition experimentally in connection with studies on starches. We do find that we get sugar in the urine of dogs under certain conditions.

Dr. J. V. Lacroix: If for no other reason, this paper is worth while because it deals with several cases in which sugar was found in the urine of dogs. While I am about it, I would suggest, by way of encouragement to those who are not doing much urinalysis, that the indican test yields much information on the subject discussed yesterday, namely, auto-intoxication due to the absorption of proteins.

Dr. H. J. Milks: Sugar did not appear in the urine of all of these cases while we were treating them. In some of the cases we did not have an opportunity to do anything in the way of treating disorders of the skin. In one case the symptoms exhibited fairly paralleled what was our idea of diabetes at that time. In one case the heart action was bad. I did not come here to discuss diabetes. You men working in the laboratory should know a lot more about it than we do in the clinic. We do encounter cases that puzzle us. When they do not respond to treatment as we think they should, naturally we wonder if there is not something else the matter with these animals. That is the gist of this paper.

STUDIES ON CANINE DISTEMPER:

II. The Effect of Injection of Filtrates of Suspensions of Dog Spleens into Ferrets*

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INTRODUCTION

In a previous communication[1] the bacteriology of the internal organs of dogs naturally infected with canine distemper was reported. A study of over one hundred affected animals indicated that *Bacillus bronchisepticus* was the etiological agent in the majority of these cases clinically diagnosed as canine distemper. In a few cases a hemolytic streptococcus was considered to have been the causative factor, while in a lesser number, *Staphylococcus albus*, thought to have been of no primary etiological importance, was found to predominate. In a very small group, an organism of the colon-typhoid group predominated, but these cases were either moribund or had died of the disease when they they were taken for autopsy and culturing.

These results were comparable with those reported by Ferry,[2,3] M'Gowan,[4] Torrey and Rahe,[5] and later by Schoiche,[6] from which these authors concluded that *B. bronchisepticus* was the etiological factor of canine distemper. These conclusions were at variance with those of Carré,[7,8] Lignieres,[9] Puntoni,[10] and Dunkin and Laidlaw,[11,12] who consider a filtrable virus to be the infecting agent.

In the examination of these naturally infected cases, particular attention was given to the bacteriology of the various internal organs, and, at the same time, experiments were undertaken to determine whether or not a filtrable virus also was present. This report covers the results of these latter experiments.

METHODS AND MATERIALS

Laidlaw and Dunkin's[13] results with filtrates of serous discharges were contrary to those of Carré[7] who based his conclusions as to the etiological virus on the infectivity of these secretions made free from bacterial contamination by filtration. However, they were able to demonstrate, without difficulty, the presence of virus in the filtrates of spleen suspensions. With the

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results of this work in mind, the spleen was selected as the tissue which would be most suitable for use in determination of the presence of a filtrable virus.

After cultures had been made from the various internal organs of the dogs being studied, the spleens were removed with aseptic precautions, weighed and ground finely with sterile quartz sand. Sufficient sterile 0.85 per cent sodium chlorid solution was added to this ground tissue to make a 20 per cent suspension. This suspension, together with the sand used in grinding the tissue, was then shaken in a mechanical shaker for 30 minutes to insure thorough disintegration of the spleen pulp. The connective tissue and cellular débris were removed by passing the suspension through a very thin layer of asbestos fiber, the reaction of which had been adjusted to pH 6.2-6.6. This reaction was comparable to those of the majority of these spleen suspensions. The clarified liquid was then filtered through No. 8 Mandler candles. Filtration through these candles was accomplished by using a low negative pressure at the beginning, increased slowly to a maximum of 13 pounds. In every instance filtration, which was carried on at room temperature, was completed within 20 to 30 minutes.

Sterility tests on these filtrates were made by seeding two fermentation tubes each containing 20 cc of 0.2 per cent glucose bouillon with 0.5 cc and 1.0 cc, respectively. At the same time an agar slant also was seeded with 0.1 cc of the filtrate under test. These cultures were incubated at 37° C. for at least 72 hours, to insure freedom from bacterial contamination. While cultures were being held for determination of sterility, the filtrate was stored at 2 to 5° C. in bottles of a size which permitted only the minimum amount of surface exposure and which were stoppered tightly and sealed with gelatin.

Since Dunkin and Laidlaw[11] had found the ferret to be so extremely well adapted as a test animal for the virus of experimental dog distemper, it was decided to use this animal for injection of the filtrates of spleen suspensions, to determine the presence or absence of a filtrable virus in the cases being studied.

Realizing the necessity of clean, healthy stock for test animals, a colony of breeding ferrets was established at our biological farm some 25 miles from the laboratory. Here the breeding animals were quartered in a building, some 200 feet from any other buildings, and in which no other animals were kept. The attendant had no contact with other animals on the farm or at his home.

No difficulty was experienced in raising clean, healthy stock, but as the work progressed the demand on this breeding colony became too great for the supply. As a result, it was necessary to purchase young ferrets from a breeder whose colony was known to be free from disease. Before using these purchased animals, however, for detection of the presence of a filtrable virus in the spleens of distemper dogs, they were quarantined in another building at the biological farm for a period of at least 30 days. From time to time, as individual ferrets were needed for test purposes, they were shipped by motor truck to the laboratory where they were kept in special quarters.

In this building the ferrets were kept in individual cages constructed so that cleaning and disinfection could be done easily. The quarters were so arranged that the attendant, who came in contact with no other animals, could make a complete change of clothing and be disinfected when entering or leaving. No persons, other than the author, had access to these quarters while the ferrets were under observation. Strict cleanliness was maintained and frequent disinfection of the quarters was carried out during the time the animals were under observation.

When sterility tests on the filtered spleen suspensions had been completed, 2 cc of the filtrate to be tested was injected subcutaneously into a healthy ferret. Since, in every case, the spleens had been made into a 20 per cent suspension, this dose of 2 cc represented the extract from 0.5 gm. of the original material. All ferrets were observed daily for any symptoms which might occur as a result of injection of an infecting agent.

Results of Injection of Filtrates Into Ferrets

For the sake of brevity, the results obtained following the injection of the sterile filtrates of suspensions of dog spleens into the ferrets have been summarized in tabular form. In table I are shown the predominating organisms recovered from the trachea, lung, liver, spleen and heart-blood of the dogs studied, as well as the effect of the injections of the spleen filtrates into ferrets.

The ferrets were held under observation for varying periods of time, depending somewhat on the availability of suitable cases of distemper in dogs for study. When cases were readily available it was found necessary to discard the ferrets before a long observation period was obtained. In every case, however, these animals were held a sufficient length of time for symptoms of the filtrable virus disease to have developed, provided this fil-

TABLE I—*Results in ferrets following injection of sterile filtrates of spleen suspensions from distemper dogs.*

DOG	PREDOMINATING ORGANISMS	EFFECT OF FILTRATE ON FERRET	REMARKS
D-1	B. B., Str.	None injected	
D-2	B. B.	Normal, 30 days	
D-3	B. B.	Normal, 30 days	
D-4	B. B.	Normal, 13 days	Died 4 days later, of B. B. infection
D-5	S. A.	Normal, 26 days	Died later, of B. B. infection
D-6	B. B.	Normal, 23 days	
D-7	B. B.	Died 2 days after injection	A ferret injected with filtrate of spleen suspension from first was normal for 30 days
D-8	B. B.	Died 21 days after injection, of B. B. infection	A ferret injected with filtrate of spleen suspension from first was normal for 30 days
D-9	S. A.	Normal, 20 days	
D-10	B. B.	None injected	
D-11	B. B.	Normal, 44 days	
D-12	B. B.	Normal, 25 days	
D-13	B. B.	Normal, 27 days. Died on 39th day, bronchopneumonia	A ferret injected with filtrate of spleen suspension from first was normal for 30 days
D-14	B. B., S. A.	Normal, 32 days	
D-15	B. B.	Normal, 26 days	
D-16	B. B.	Normal, 22 days	
D-17	B. B.	Normal, 32 days	
D-18	B. B.	Normal, 30 days	
D-19	B. B., S. A.	Normal, 52 days	
D-20	B. B.	Normal, 40 days	
D-21	B. B.	Normal, 77 days	
D-22	B. B.	Normal, 44 days	
D-23	B. B.	Normal, 44 days	
D-24	B. B., S. A.	Normal, 44 days	
D-25	B. B., S. A.	Normal, 77 days	
D-26	B. B.	Normal, 31 days	
D-27	B. B.	Normal, 32 days	
D-28	B. B.	Normal, 30 days	
D-29	B. B.	Normal, 30 days	
D-30	B. B.	Normal, 30 days	
D-31	B. B., S. A.	Normal, 26 days	Serous nasal discharge on 30th day. Destroyed. Filtrate of spleen suspension into another ferret, which remained normal for 30 days
D-32	B. B.	Normal, 31 days	
D-33	B. B.	Normal, 19 days	Diarrhea and serous nasal discharge on 20th day. Destroyed. Filtrate of spleen suspension to another ferret, which remained normal for 30 days
D-34	B. B., Str.	Normal, 30 days	
D-35	B. B.	Normal, 30 days	
D-36	B. B.	Normal, 30 days	
D-37	B. B.	Normal, 28 days	

TABLE I—*Results in ferrets following injection of sterile filtrates of spleen suspensions from distemper dogs—Continued.*

DOG	PREDOMINATING ORGANISMS	EFFECT OF FILTRATE ON FERRET	REMARKS
D-38	B. B.	Normal, 28 days	Destroyed 30th day, strep. pneumonia
D-39	S. A.	Normal, 30 days	
D-40	B. B.	Normal, 31 days	
D-41	B. B.	Normal, 30 days	
D-42	B. B., S. A.	Normal, 30 days	
D-43	B. B.	Normal, 32 days	
D-44	B. B., Str.	Normal, 30 days	
D-45	B. B.	Normal, 31 days	
D-46	B. B.	Normal, 31 days	
D-47	B. B., S. A.	Normal, 30 days	
D-48	S. A.	Normal, 32 days	
D-49	B. B.	Normal, 30 days	
D-50	B. B., C. T.	Normal, 22 days	Died 10 days later, bronchopneumonia
D-51	B. B.	Normal, 33 days	
D-52	C. T.	Normal, 25 days	Destroyed 3 days later. Bronchopneumonia due to B. B.
D-53	B. B.	Normal, 21 days	Destroyed 5 days later, strep. pneumonia
D-54	B. B., S. A.	Normal, 13 days	Died 4 days later, strep. pneumonia
D-55	B. B.	Normal, 14 days	Died 4 days later, strep. pneumonia
D-56	B. B.	Normal, 14 days	Died 4 days later, strep. pneumonia
D-57	B. B., S. A.	Normal, 23 days	Died 6 days later, B. B. infection
D-58	B. B.	Normal, 23 days	Died 6 days later, B. B. infection
D-59	B. B.	Normal, 23 days	Died 6 days later, B. B. and strep. infections
D-60	S. A., Str.	Normal, 21 days	Died 5 days later, B. B. infection
D-61	B. B.	Normal, 27 days	
D-62	B. B.	None injected	
D-63	B. B.	None injected	
D-64	B. B., S. A.	None injected	
D-65	B. B., S. A.	None injected	
D-66	B. B.	Normal, 13 days	Destroyed 2 days later, B. B. pneumonia
D-67	B. B.	Normal, 21 days	Destroyed 3 days later, strep. pneumonia
D-68	B. B.	Normal, 22 days	
D-69	B. B., S. A.	Normal, 16 days	Died 7 days later, bronchopneumonia
D-70	B. B.	None injected	
D-71	B. B., S. A.	Normal, 30 days	
D-72	None*	None injected	
D-73	None*	None injected	
D-74	B. B.	None injected	
D-75	Str.	None injected	
D-76	B. B., C. T.	None injected	
D-77	B. B., C. T.	None injected	

TABLE I—*Results in ferrets following injection of sterile filtrates of spleen suspensions from distemper dogs—Concluded.*

DOG	PREDOMINATING ORGANISMS	EFFECT OF FILTRATE ON FERRET	REMARKS
D-78	C. T.	None injected	
D-79	C. T.	None injected	
D-80	B. B., S. A.	Normal, 16 days	Died 5 days later, strep. infection
D-81	None*	None injected	
D-82	None*	None injected	
D-83	B. B.	None injected	
D-84	B. B., S. A.	None injected	
D-85	Str.	Normal, 31 days	Found dead on 31st day. Autopsy negative
D-86	B. B.	Normal, 23 days	Died on 30th day. Cultures negative
D-87	B. B.	Normal, 18 days	Died on 29th day, strep. pneumonia
D-88	B. B.	Normal, 14 days	Died 5 days later. Cultures negative
D-89	B. B.	Normal, 30 days	
D-90	B. B.	None injected	
D-91	Str.	Normal, 20 days	Died 9 da ys later, strep. infection
D-92	None*	None injected	
D-93	Str.	Normal, 12 days	Contracted infection from fitch. Recovered. See text
D-94	S. A., Str.	None injected	
D-95	Str.	None injected	
D-96	Str.	Normal, 22 days	
D-97	Str., S. A.	Normal, 20 days	This ferret injected with 50 mg. dog spleen
D-98	S. A.	Normal, 16 days	This ferret injected with 50 mg. dog spleen. Died 8 days later, strep. infection. A ferret injected with 0.5 gm. spleen was normal for 20 days
D-99	Str., S. A.	None injected	
D-100	B. B., Str.	None injected	
D-101	B. B.	Normal, 15 days	Injected with 40 mg. dog spleen
D-102	B. B.	Normal, 15 days	Injected with 40 mg. dog spleen
D-103	B. B., S. A.	Normal, 15 days	Injected with 40 mg. dog spleen
D-104	B. B., C. T.	Died of septicemia, 48 hours after injection of 40 mg. dried dog spleen	
D-105	B. B.	Died of septicemia, 48 hours after injection of 40 mg. dried dog spleen	

*Normal dog.
B. B. = *Bacillus bronchisepticus.*
Str. = Streptococcus.
S. A. = *Staphylococcus albus.*
C. T. = Colon-typhoid organism.

trable virus was contained in the filtrates of spleen pulp. Dunkin and Laidlaw[11] found this incubation period to be about ten days, which later work with virus kindly supplied by Major Dunkin proved to be the case.

Filtrates of suspensions of spleens from the dogs were not injected in some few cases either because the dogs being studied were normal, in which case no apparent advantage was to be had by such injection, or because the ferrets on hand for use had not been quarantined a sufficient length of time when the diseased dogs were available for study.

In a few cases (D-97 and others) filtrates of suspensions of the dog spleens were not injected, but instead massive doses of unfiltered spleen suspensions were used. Regardless of whether the ferrets were injected with the filtered or the unfiltered suspensions of distemper dog spleens, the symptoms resulting from filtrable virus infection, described in detail by Dunkin and Laidlaw,[11] were not observed in the ferrets used.

In a few instances, in spite of the quarantine precautions, there were outbreaks of infection in the animals under observation but bacteriological studies and further tests on ferrets indicated that they were of bacterial origin.

Discussion

In carrying out these experiments the dosage of the filtrates (2 cc representing the extract of 0.5 gm.) of the spleen suspensions was deemed adequate, since Laidlaw and Dunkin[13] had found that 0.001 gm., and in some cases 0.0001 gm., of the infective spleen was sufficient to produce typical symptoms in the ferret within nine or ten days. In their experiments on the filtrability of the infectious agent, they found that 2 cc of a filtrate of a 5 per cent suspension (representing 0.1 gm. of spleen pulp) was sufficient to produce typical symptoms in twelve days.

While every precaution was taken to prevent spread of any infection to the ferrets under observation, yet by some means such infection did gain entrance, with the result that two outbreaks of streptococcus and *B. bronchisepticus* infection were experienced. That these infections were primary and not secondary to filtrable virus infection is shown by the fact that filtrates of suspensions of spleens of these animals caused no apparent reaction in other ferrets during the 30 days they were under observation. The outbreaks did show, however, that ferrets are very susceptible to infection by streptococci and *B. bronchisepticus*.

Since the result of the work indicated that a filtrable virus was not present in the spleens of the distemper dogs studied, it was thought advisable, in the last few experiments, to determine the effect of injection of unfiltered distemper dog spleens on ferrets (D-97 and others). In these cases the spleens were ground finely in sterile 0.85 per cent saline solution and rather massive doses (40-50 mg.) injected subcutaneously.

One animal (F-D-98) died on the twenty-fourth day, following an eight-day illness from streptococcus infection, which was not transmitted to another ferret by the injection of 0.5 gm. of spleen. This second ferret later contracted a disease, like distemper in the ferret, from an affected fitch which was being studied. Another ferret (F-D-93) also contracted this disease of the fitch but recovered. A report of this outbreak will be made in a later publication.

All other ferrets except the last two (F-D-104, F-D-105) were normal during the fifteen days of observation, a period greater than necessary for the development of filtrable virus symptoms.

The last two ferrets (F-D-104, F-D-105) died as a result of generalized septicemia within 48 hours after the injection of the suspensions of dried dog spleens. Septicemia was no doubt due to contaminating organisms present in the dried spleen.

SUMMARY

Subcutaneous injection of ferrets with sterile filtrates of suspensions of spleens from dogs naturally infected with distemper did not produce symptoms of the filtrable virus dog distemper in these test animals.

Similar negative results were obtained in the few instances tested, following the injection of unfiltered spleen suspensions of dog spleens.

While a few of the ferrets became infected with streptococci and *B. bronchisepticus*, it was evident that these bacterial infections were primary and not secondary to filtrable virus infection, since the disease could not be transmitted to other ferrets by subcutaneous injection of sterile filtrates of or unfiltered suspensions of spleens from the affected animals.

CONCLUSIONS

The results obtained following the injection of sterile filtrates of suspensions of spleens from distemper dogs into ferrets indicate that a filtrable virus was not an etiological factor in the naturally infected cases of dog distemper studied, from the majority of which *Bacillus bronchisepticus* had been recovered.

Symptoms of filtrable virus infection did not occur in ferrets following the injection of massive doses of unfiltered suspensions of spleens taken from a few naturally infected cases.

Since by the use of ferrets as test animals the presence of a filtrable virus was not demonstrated to have been present in the naturally infected cases studied and since *B. bronchisepticus* was recovered from the majority of these naturally infected cases, this organism should be considered as of primary etiological importance in the symptom complex diagnosed clinically as canine distemper. Likewise, streptococci and possibly organisms of the colon-typhoid group also should be so considered in some few cases.

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An Excellent Choice

Congratulations to the members of the A. V. M. A. on their excellent judgment in choosing Dr. N. F. Williams, Fort Worth, Tex., as President. As was predicted in the May-June *Norden News*, his nomination met with unanimous approval and thus the Association is assured of a worthy successor to Dr. R. R. Dykstra, who has served so ably in the term just concluded.

Dr. Williams will need full measure of coöperation from every veterinarian this year to surmount the difficulties arising from the economic situation. You, who are not members of the Association, join hands with those who are, and let us all work for the betterment of the organization, and thus the individual.

The Norden News.

TRICHOMONIASIS IN CATTLE

A Preliminary Report*

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INTRODUCTION

Protozoan infections of domestic animals have not received proper recognition by veterinarians in the past. The majority of the mammalian protozoan diseases, regardless of pathogenicity, have been studied and reported by protozoölogists, zoölogists and physicians. It is hoped that the following report will stimulate veterinary investigators to make a more concerted effort in the field of protozoölogy.

The existence of protozoan invasions of the genital tract of cattle, to the best of my knowledge, has not been reported in this country. There is much in the literature regarding vaginal trichomoniasis in women, but such a disease in cattle or one simulating the condition in the human apparently has been a nonentity. Its occurrence was first manifested to me when I had occasion to examine several dairy sires for impotency. Failing to find anything abnormal in these bulls, I asked to examine some of the cows bred to these sires. One cow (86) had received service two months previously and had not come in heat since. Another cow (163) had received service a few days over a month previously and was believed to have conceived.

Cow 86 was examined with the vaginal speculum first. Upon insertion of the speculum, a peculiar, whitish-colored, odorless, semi-watery, mucous-like fluid was observed to flow from it. During the act of collecting some of this fluid in a test-tube, a thick, semi-transparent clump of mucous, thought to be the cervical seal, was observed to drop from the vaginal speculum. The test-tube containing the sample was placed in a vest pocket close to the body in an attempt to keep it warm until the laboratory was reached. Cow 163 was examined the same day and a cervical seal was visible. At a later date, she was examined again and a very similar discharge and a thick clump of mucous, as described for cow 86, was collected.

The following day, a fresh smear was made from the sample obtained from cow 86. This was examined under low power and in a darkened field obtained by lowering the condenser from the

*Presented at a meeting of the Schuylkill Valley Veterinary Club, Pottsville, Pa., April 20, 1932.

microscopic stage. Motile flagellates with undulating membranes were observed swimming about in the field. Closer observation revealed myriads of the protozoan flagellates in this one smear. Further study proved these unicellular structures to belong to the genus Trichomonas.

Classification according to Wenyon[1]:

Phylum: Protozoa
Sub-phylum: Plasmodroma
Class: Mastigophora
Sub-class: Zoomastigina (monozoic forms)
Order: Protomonadida
Sub-order: Eumonadea
Family: Trichomonadidae
Genus: Trichomonas

This identification of the protozoan was confirmed by Dr. David H. Wenrich, protozoölogist in the Department of Zoölogy, University of Pennsylvania.

Review of Literature

Mazzanti[2] (1900) is given credit by European workers as having been the first to discover trichomonads in cattle. Apparently, little appeared again in veterinary literature regarding trichomonad infection in cattle until 1925, when Drescher[3] cited Hofengärtner as having found them in the stomach fluid of a seven-month-old fetus. Pfenninger,[4] working independently, confirmed Hofengärtner in a paper which he published in 1927. Riedmüller,[5, 6] in papers presented in 1928 and 1929, probably has given us our most comprehensive and complete knowledge of trichomoniasis as it relates to veterinary medicine. He believes trichomoniasis to be a causative agent of abortions and classifies it as one of an infectious nature. I think it might be well to list here the causes of abortions, both infectious and non-infectious, as given by Riedmüller,[6] since we are apt to lose sight of causative factors other than *B. abortus* in this day of abortion control work. Quoting from Riedmüller:

> The causes of non-infectious abortion may be of the following nature: (1) mechanical or traumatic; (2) disturbances of metabolism: (a) overstrain, want of light, illness, (b) nutritive deviation (lack of iodin); (3) cold; (4) psychical influence; (5) poisons. As causes of infectious abortion we may consider: (1) Coccaceae (diplococci, streptococci); (2) Bacteriaceae (a) *Corynebacterium abortus* (Bang); (b) *B. coli, B. paratyphus, B. enteritidis* (Gärtner) (proteus); (c) *B. pyogenes bovis;* (d) *Bact. tuberculosis;* (3) vibrios, spirilla; (4) Hyphomycetae; (5) protozoa.

Returning to Mazzanti, the three cases which he observed were in mature cattle which remained permanently sterile and

he believed this to be caused by the trichomonads. Thus we have the possibility of sterility resulting from trichomoniasis.

Hofengärtner, Pfenninger[4] and Riedmüller,[5, 6] have all reported the finding of trichomonads in fetuses. The two latter authors have found the protozoa in the fluid stomach contents, intestinal contents, peritoneal and pleural transudates and in the pericardial fluid.

Riedmüller has been successful in inoculating pregnant guinea pigs intraperitoneally, producing abortion and sometimes death. Only in one aborted guinea-pig fetus was he able to demonstrate trichomonads. He states that the diplococci associated with the flagellates proved to be non-pathogenic for susceptible guinea pigs when pure cultures were injected subcutaneously and intraperitoneally. In guinea pigs, he has been able to recover trichomonads from the pleural and peritoneal fluids, from the pericardial fluid, the uterus and occasionally the subcutis. Riedmüller states that it is very difficult to obtain successful "takes" following intraperitoneal injections and that it was only after seven guinea pigs were injected that he finally was successful. The writer has injected only one guinea pig, which later aborted, and was unable to demonstrate trichomonads in either the fetuses (four) or the mother.

Vital Morphology

It is not my intention to give an accurate and complete description of the morphology of these protozoa. Such will be given in a later paper by a protozoölogist. Suffice it to say here that the trichomonad is slightly larger than a leukocyte and smaller than an epithelial cell found in a vaginal smear. They assume many shapes and forms, ranging from pyriform and fusiform to oviform or roundish. Three flagella are usually observed protruding from the anterior end or blepharoplast. These flagella are as long if not slightly longer than the body. Originating from or near the blepharoplast is seen the undulating membrane. This membrane extends the entire length of the body and when the form of the body is changed from pyriform to oviform, it becomes longer than the body. A trailing flagellum is noted incorporated in the attached border of the undulating membrane which extends beyond its posterior end. The nucleus is large and is most often located in the anterior portion of the cell. The motility of the organism seems to vary. The flagella are seen to be extended in front of the cell and then vigorously lashed back against the side of the body. Thus, a forward move-

ment is manifested. Some of the microörganisms move with a spiral motion of the body, the flagella being seen first on one side and then on the other. The undulating membrane seems to be in a constant state of agitation. Cold seems to prolong the life of these organisms rather than kill them. One test-tube sample kept at room temperature remained alive for eleven days, and another for nineteen days, whereas a sample collected in physiological salt solution and kept in the incubator at 37.5° C. remained alive only three days.

Staining Properties

Trichomonads fail to stain satisfactorily when the ordinary stains are used. This may account for the failure of earlier recognition of this type of invasion of the genital organs of cattle and should emphasize the importance of making microscopical examinations of fresh smears.

Clinical Symptoms

Very little in the way of external visible symptoms are manifested by an animal with trichomoniasis. One often observes a collection of dried mucous and manure on the tail and vulvar hairs. When an animal lies down, one will usually see a mucopurulent material being exuded from the vulva into the gutter. One diagnosis of trichomoniasis was made by walking along behind a group of stabled animals and observing a collection of mucopurulent material in the gutter. This animal (163) proved to be positive when the discharges were observed under the microscope.

The mucopurulent discharge mentioned above is peculiar to trichomoniasis. It is made up of clear or slightly cloudy mucus, incorporated in which are clumps of a cream-colored pus. When this pus is examined, trichomonads are usually found. When an animal is in heat, these clumps of pus may not manifest themselves but the mucous discharges will appear cloudy and somewhat watery. There is no distinguishable odor to the discharge and every sample that the writer has examined has been alkaline in reaction to litmus. This is not in accord with the reports of Mazzanti[2] or with the findings in the human.[7] All cows examined that proved to be positive to trichomoniasis have had a granular vaginitis.

If a cow has remained standing for some time prior to examination with the vaginal speculum, one will usually observe a collection of mucous or mucopurulent material on the floor of the vagina anterior to the posterior sphincter and just below the

cervix. Occasionally, one will see the mucopurulent material being exuded from the cervix. One very seldom sees any cervicitis or vaginitis in these cases. Usually, however, the vestibule and vulva appear inflamed, but whether or not this is caused by the trichomonads, granular vaginitis or other organisms, the writer is unable to say. Small ecchymotic hemorrhages have been observed on the cervix and anterior portion of the vaginal wall.

Estrum seems to be disturbed in animals showing trichomonads in that it occurs irregularly or the diestrual period is greatly prolonged.

Transmissibility

A normal cow (A) was observed several times prior to artificial inoculation. Her discharges proved to be free of trichmonads. On February 20, 5 cc of secretions containing trichomonads taken from cow 86 were deposited in the vagina of the above-mentioned normal cow A. Previous to introduction, the secretions to be used were examined under the microscope and were found to contain numerous very active trichomonads. Cow A was examined on the fourth and ninth days following introduction of the secretions and was negative. On the tenth day, she became positive and remained so for twenty-five days. Since that time she has remained negative.

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Monkey Commits Suicide at Zoo

Is the depression causing animal suicides as well as human? A monkey in an English zoo recently committed suicide, apparently deliberately. Sightseers watched him gnaw off a six-foot piece of rope that was hanging in his open-air cage. He attached one end of the rope to the bough of a tree in the cage. Then he made the other end into a noose, into which he inserted his head. He pulled the slip-knot tight, climbed as high as the rope would allow, and jumped. Death was instantaneous.

FEEDING OF THE BRUCELLA ORGANISM TO CHICKENS AND ITS EFFECT ON EGG-PRODUCTION OF PULLETS AND ON GROWTH OF YOUNG CHICKS*

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Since our previous reports on the effect of the Brucella group of organisms on chickens, other investigations have been reported. Gilman and Brunett[1] found some naturally infected chickens in New York State, where the condition was neither serious nor widespread, and there was little or nothing to indicate that egg-production was influenced. Injection caused a higher, more lasting agglutination titre than feeding. In exceptional cases, infected birds retained the organism for a considerable time, in one instance for 138 days after injection. Strange and Beach[2] injected and fed chickens with various types of Brucella from different sources without producing clinical evidence of infection and without finding the organism when the birds were autopsied.

In our previous work, experimental feeding of Brucella did not produce symptoms or gross lesions of infection. Injection produced similar results except that large doses caused temporary depression. Artificial inoculation and feeding having failed to cause significant disease, an attempt was made to find naturally infected birds. Examination of several thousand chickens revealed a few birds with agglutinins, but without symptoms or gross lesions. This work failed to give any measure of the injury caused when chickens eat infective material. Variations in egg-production in hens and in growth of young chickens are delicate tests and were next employed to determine the amount of damage resulting from feeding Brucella cultures. Data from these experiments furnish the material for the following report.

Twenty high-production White Leghorn pullets were purchased October 21, 1930, and placed in a 12x16 poultry-house. Individual egg records were kept from October 21, 1930, until May 24, 1931. Total daily egg-production was recorded until June 25, when the birds were killed for final examination. Check chickens consisted of 174 pullets in the same flock from which the 20

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experiment birds were obtained. The highest egg-production of laying hens in this geographical section is in April. Hence on April 9, ten large, heavily grown, slant-agar cultures were fed in 1000 cc of water. Each bird received enough culture to make at least 15 cc of suspension corresponding in density to tube 2 of the McFarland nephelometer. The contaminated water was consumed in two hours. Cultures fed were two highly virulent porcine strains of recent isolation.

In the accompanying graph the egg-production both before and after feeding is represented and compared with that of the check lot of chickens. Percentage computations are made on five-day averages. The infected lot showed a higher egg-production than the check lot before feeding Brucella (April 1 to 9) and also for 8 days (April 9 to 17) following feeding. Then for a period of 6 days (April 17 to 22) there was a slight slump, lower than the check lot, followed by an increase, so that at the end of the month the infected lot were producing more heavily than the unfed checks. Whether or not these slight variations in production are significant is questionable. The decreased production from April 17 to 22 inclusive is suggestive.

The chickens were fed on a very laxative ration throughout the entire experiment, so that all showed a slight diarrhea. Their only change in condition following feeding was a considerable increase in looseness of the bowels for a few days. A laxative effect of feeding cultures has not been observed in any of our other experiments. However, in no other experiment have the birds been on a diet laxative enough to produce a near-diarrhea. The chickens remained on experiment until June 25, when they were killed. Production throughout had been satisfactory. On autopsy no lesions attributable to Brucella infection were observed. One chicken showed extensive lesions of range paralysis and twelve showed some inflammation of one or both feet ("bumble foot"). From the foot lesions staphylococci and *Esch. coli* were isolated.

In the second experiment an attempt was made to determine the influence of feeding Brucella cultures on the growth of young chicks. One hundred and two White Leghorn chicks 10 days old were divided into two equal lots, one being fed Brucella cultures and the other serving as checks. Since our stock cultures did not contain strains of Brucella significantly pathogenic for chickens, or strains that had been isolated from farm flocks, an attempt was made to develop a more pathogenic strain. For this a stock culture was selected that produced a good agglutina-

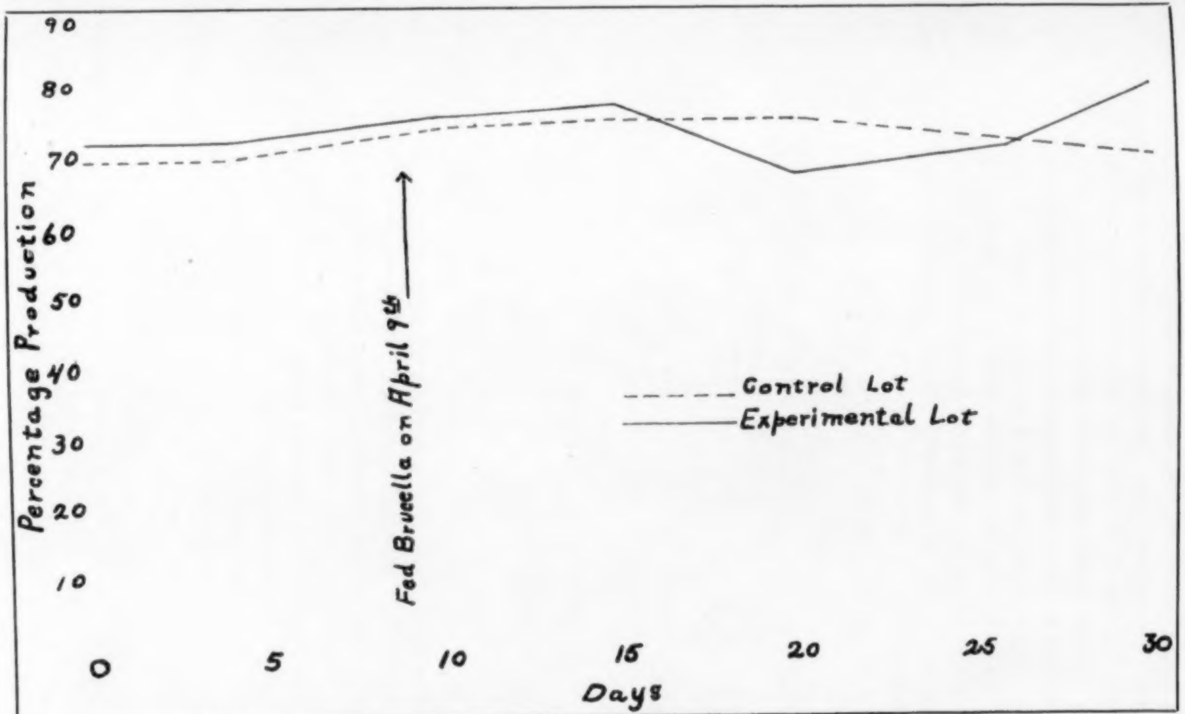

FIG. 1. Graphic chart showing egg-production of pullets fed Brucella experimentally, compared with egg-production of control lot.

tion titre in chickens. Its injection into chickens resulted in a high agglutination titre, shortly after which the birds were killed, the organism was reisolated, and the procedure was repeated. A culture that had been passed through chickens seven times was used to feed the young chicks in this experiment. Since it is conceivable that feeding any non-pathogenic microörganism in high concentration to very young chicks may produce unfavorable results, a strain of *Esch. coli* non-pathogenic for chickens was fed to the check lot at the first five feedings in quantities equal to the Brucella fed the infected lot. The cultures were fed in drinking water. Table I shows the results of feeding.

TABLE I—*The growth of chicks fed Brucella compared with that of controls.*

ORGANISM FED	DATE (1931)	AMOUNT FED	CONDITION OF CHICKS	AVERAGE WEIGHT
Check lot of 51 chicks, fed cultures of *Esch. coli*	7- 2	1 culture	Good	
	7- 7	1 culture	Good	
	7- 9	1 culture	Good	
	7-14	2 cultures	Good	
	7-15	4 cultures	Good	
	7-29	Nothing	Good	
	8-18	Nothing	Good	0.84 lbs.
Lot of 51 chicks, same weight and age as above, fed cultures of *Br. abortus*	7- 2	1 culture	Good	
	7- 7	1 culture	Good	
	7- 9	1 culture	Good	
	7-14	2 cultures	Good	
	7-15	4 cultures	Good	
	7-29	11 cultures	Good	
	8-17	Nothing	Good	0.85 lbs.

The chicks were fed a limited amount of culture until it was observed that this had no effect. Then they were fed increasingly larger amounts until 27 days later, when they were given their maximum dose, more than one slant-agar culture to 5 chicks. Feeding produced no symptoms, did not influence growth, the infected lot averaging an insignificantly (0.01 lb.) greater weight than the check lot at the termination of the experiment. Neither lot showed rapid growth but both check and infected lots were healthy during the entire experiment.

CONCLUSIONS

Feeding of Brucella cultures to laying pullets was followed by a temporary, slight decrease in egg-production.

Feeding of Brucella to 10-day-old chicks had no effect.

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OBSERVATIONS ON THE PROSTATE GLAND OF THE DOG*

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The prostate gland of the dog, as observed at necropsy made as a routine, varies greatly in size. There is no apparent relationship between the size of this organ and the weight of the body. Benign types of lesions are common. Because of these observations it was thought of interest to obtain data with regard to weight and size of the average normal prostate gland.

The prostate glands of 120 dogs were obtained for gross and microscopic study. Lesions were found in a high percentage of cases. The previous environment and approximate age of each dog was noted, and certain factors influencing the growth and development of the prostate gland were considered.

The dogs from which the glands were taken were aged from one to fifteen years. Several breeds were represented, the mongrel shepherd predominating. Because of the possible effect of environment on the gland, the animals were grouped as follows: group 1, house-trained dogs; group 2, kennel-raised dogs, and group 3, miscellaneous dogs, most of which were farm-raised. Some of the latter group, perhaps, could have been included in group 1, as they were accustomed to sleeping indoors in cold weather.

The glands were weighed while fresh. They were then fixed in a 10 per cent solution of formalin and cross-sectioned into pieces 0.5 cm. thick. All sections were studied macroscopically and microscopically. Forty-eight (40 per cent) were found to be essentially normal histologically and 72 (60 per cent) were classified as pathologic.

NORMAL PROSTATE GLANDS

Grossly the prostate glands appeared smooth, dense and symmetrically bilobed. They were small, weighing 0.8 to 8.1 gm. each. The mean weight was 3.9 gm. and the standard deviation of the weight was 2 gm. Seventeen per cent fell below 2 gm., 66 per cent weighed 2 to 6 gm., and the remaining 17 per cent more than 6 gm. each. There was no significant relationship between prostate gland and body weight. The coefficient of varia-

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tion, which usually is about 10 to 20 per cent, comes out 35 per cent for weight of body and 53 per cent for weight of gland. The correlation coefficient of weight of body and weight of gland was extremely low (0.12). This indicates that one cannot closely estimate the weights of the glands from the weights of the bodies of the dogs in this series. Since 8.1 gm. represents the maximal weight observed in the normal gland, it will be referred to in later comparisons.

Histologically an abundance of smooth muscle could be demonstrated throughout the capsule and stroma. The comparative relationship of stroma and epithelial tissue to acinar spaces depended somewhat on the age of the dog. In the young animal the acini were small and of irregular size. They appeared to increase in size with the age of the animal. The acini were lined chiefly with a single layer of cuboidal cells; however, occasional small areas of columnar cells were present in some. The cell nucleus was situated near the base of the cell. It was rather large and appeared granular. The walls of the vessels were normal in thickness. Corpora amylacea were noted in two glands. These were obtained from aged dogs.

Abnormal Prostate Glands

Seventy-two prostate glands were classified as pathologic. They weighed 1.5 to 260 gm. each. Forty-nine weighed more than 8.1 gm., the maximal weight observed in the normal series. Thirty-eight of the glands were obtained from dogs six years or more of age.

Grossly some of the glands appeared normal. However, the greater number of them were nodular and irregular of shape. On cross-section, adenomas and cysts were observed in many. The cysts varied greatly in size and contained colloid-like material or clear fluid. One large gland contained 200 gm. of clear cystic fluid. Abscesses were not grossly visible.

Microscopically, an increase in quantity of stroma was noted in six glands. All were obtained from aged dogs, four of which had been castrated. The acini in these glands were diminished in size and in many areas entirely obliterated. The remaining sixty-six glands all revealed marked adenomatous hypertrophy. The acinar cells were high columnar in type. Reduplication of cells and papillomatous infoldings were common. The acini varied greatly in size. Many were markedly dilated and coalescing. In ten, cystic degeneration was so marked as to be visible macroscopically. These were later diagnosed as papillary cystadenomas. So-called corpora amylacea were observed in twenty

of the glands. All were from aged dogs. Round cell and leucocytic infiltrations were observed in some. Thickening of the walls of the vessels and thrombosis were common.

Factors Influencing the Size of the Prostate Gland

Group 1: Three of the seventeen house-trained dogs in this group had been castrated; they were aged from ten to fourteen years. The remaining fourteen males were aged from one to fourteen years. The prostate glands of the entire group were histologically abnormal.

The prostate glands of the fourteen animals that had not been castrated varied greatly in size. They weighed from 1.5 to 160 gm. Twelve (85 per cent) weighed more than 8.1 gm., eight of which weighed more than 45 gm. each. The two glands that weighed less than 8.1 gm. each were both obtained from young dogs. Marked adenomatous hypertrophy was observed in all. Cystic degeneration was observed in three. Corpora amylacea were common.

The prostate glands of the three castrated dogs were small and firm. They weighed 1.5, 3.4 and 4.3 gm. each. The largest gland was obtained from a dog that had been castrated at the age of five years. Atrophy of the cells lining the acini was revealed microscopically. The acini were greatly diminished in size and number. Fibrous tissue predominated. The two other dogs had been castrated early in life. There was an almost complete absence of acini in these glands. Adenomatous development had failed entirely.

Group 2: The twenty-eight dogs in this group had been in kennels for one year or more each. Fourteen were definitely aged, the others were adults aged more than one year. The habits of these dogs differed from those of group 1 in that they were at liberty to urinate at their convenience. They differed from those of group 3 in that they were not constantly urged to urinate by observance of posts or other objects.

The prostate glands of this group weighed from 2.2 to 75 gm. each. Twenty-one glands (75 per cent) were abnormal and only seven (25 per cent) were classified as histologically normal; one gland was from an aged dog. Sixteen glands (57 per cent) weighed 8.1 gm. or more each, of which only one weighed more than 20 gm. Twelve glands weighed less than 8.1 gm. each. Twenty-one glands revealed marked adenomatous hypertrophy. Thirteen of these were from aged dogs. Cystic degeneration was not observed. Corpora amylacea were common.

Group 3: There were seventy-five miscellaneous and farm-raised dogs in this group. Twenty were definitely aged and the other fifty-five were aged one year or more. One of the aged dogs was castrated early in life.

The prostate glands of this group weighed 0.8 to 260 gm. each. Thirty-five (46 per cent) were abnormal and forty (53 per cent) were normal. Twenty-three (30 per cent) weighed more than 8.1 gm., ten of which weighed more than 20 gm. each; one weighing 260 gm. was a cystic gland that contained 200 gm. of fluid. Thirty-one glands revealed marked adenomatous hypertrophy; seven of these also showed marked cystic degeneration. Fibrosis was definite in four glands. One of these was from a castrated animal. Forty prostate glands in this group were classified as histologically normal. One was from an aged dog.

Reviews from the Literature

Sisson[1] stated that the prostate gland of the dog is relatively large and is subject to much variation in size.

Variations in size of the prostate gland have been observed in other animals and in man. Bell[2] mentioned that hypertrophy of the gland is the most common disease of this organ in man. In about 8 per cent of men aged more than sixty years hypertrophy is sufficiently pronounced to cause symptoms. From 10 to 15 per cent of enlarged prostate glands in man are carcinomatous. Tuberculosis and pyogenic infections account for enlargement in a small number of cases.

Fox[3] observed disease of the prostate gland in five captive wild animals. In three, hypertrophy was benign; in one animal the lesion was tuberculous, and in the other the lesion suggested neoplasia. Dollar[4] stated that hypertrophy of the prostate gland, as seen in elderly men, when occurring in animals is almost entirely confined to dogs. Feldman[5] observed enlargement of the prostate gland in only one animal other than the dog. This occurred in an old castrated boar, in which the gland was about 10 cm. in diameter. Histologically, prostatitis with fibrosis and edema was revealed. Lund,[6] French,[7] Hobday,[8] Müller,[9] and Nieberle and Cohrs[10] stated that hypertrophy of the prostate gland is common in old dogs.

Goodpasture and Wislocke[11] reported enlargement of the prostate gland in eight old dogs examined by them. Goodpasture[12] later examined fifty old dogs. Thirty-seven of these were males. Seventeen had benign tumors of the prostate gland. In two there were changes noted which may be interpreted as being of

a carcinomatous nature. He reported the individual weights of fifteen dogs. Twelve weighed 10 gm. or more each. Smith[13] studied senile changes in the testes and prostate glands of thirty-two dogs aged from six weeks to twenty years. Prostatic changes were observed at about the age of six years. Wooldridge[14] reported a prostate gland of a dog that weighed 840 gm.

Comment

The lack of correlation in weight of the normal prostate gland and weight of the body was quite definite. It was observed that small glands were as common in large dogs as in the smaller breeds. However, since the largest normal gland in this study weighed only 8.1 gm., regardless of the weight of the dog, it would appear within reason to state that any gland weighing more than 10 gm. is abnormal. This, however, could not imply that those weighing less than 10 gm. were normal. Indeed, a 1.5-gm. gland from a fox terrier aged one year microscopically revealed general adenomatous hypertrophy. It was observed that 66 per cent of all normal glands weighed from 2 to 6 gm. each.

The diseased prostate glands varied greatly in size. Weight alone was no criterion of their histologic condition. Benign adenomatous hypertrophy was the most common lesion observed. In some glands this may have been physiologic rather than pathologic. True neoplasia was not observed. However, adenomatous change undergoing cystic degeneration was common. Adenomatous hypertrophy was very common among aged dogs. In this respect the histologic behavior of the prostate gland is exactly opposite that of the thyroid gland. Since sex activity becomes quiescent with age, and atrophy of the other sex organs occurs, it would appear that hypertrophy of the prostate gland may be a purely compensatory activity as related to sex. However, this is doubtful because orchidectomy causes atrophy of the prostate gland.

It was observed that the testes are essential for the normal development and growth of the prostate gland. In three dogs in which orchidectomy had been performed early in life, the prostate gland failed to develop. In another dog on which the same operation was performed at the age of five years, marked atrophy of the adenomatous tissues of this organ was observed seven years later. Hobday[8] cited three cases in which castration was performed to relieve hypertrophy of the prostate gland; good results were obtained in all.

On viewing the incidence of lesions in the three groups of dogs, one might at first conclude that since all house dogs had

abnormal prostate glands, and that only 46 per cent of the glands from farm dogs were abnormal, environment, perhaps forced urinary retention, is of some significance. However, all but two of the house-trained dogs were aged. It was noted that lesions of the prostate gland were rather constant in all aged dogs regardless of environment. It was observed, however, that the prostate glands obtained from the dogs of group 1 were definitely much larger than those of groups 2 and 3.

Summary and Conclusions

The correlation coefficient for weight of body and weight of prostate gland is very low. One cannot estimate with any degree of accuracy the weight of a dog's prostate gland by knowing the weight of his body.

Eight and one-tenth grams was the maximal weight observed in the series of histologically normal prostate glands.

Lesions of the prostate gland are very common among dogs. Seventy (60 per cent) of the dogs that had not been castrated had lesions of the prostate gland. Forty-three of these were aged dogs.

All house-trained dogs had lesions of the prostate gland. The smallest abnormal gland was from this group. The remaining glands of this group were comparatively larger than those of groups 2 and 3.

True neoplasia was not observed. However, cystadenomatous degeneration was common.

Castration definitely arrested development of the prostate gland of the dog.

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CHRONIC CARRIERS OF INFECTIOUS LARYNGOTRACHEITIS*

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The first report of carriers in infectious laryngotracheitis was made by Gibbs[1] after examining 144 birds by means of intratracheal swabs and discovering two birds which eliminated virus longer than usual although showing no visible symptoms of disease. A further study of 504 birds, which had recovered from infectious laryngotracheitis, definitely identified 14 chronic carriers.[2] At the same time that these experiments were in progress, Komarov and Beaudette[3] were making similar studies of infectious laryngotracheitis and arrived at the same conclusion, namely, that chronic carriers were a reality in this disease and that the virus was quite generally eliminated from some portion of the larynx and trachea. Studies of infectious laryngotracheitis carriers have been continued and some new things have been learned. The object of this paper is to report the newer knowledge pertaining to infectious laryngotracheitis carriers.

The number of days that each of the 21 chronic carriers studied eliminated sufficient virus to inoculate susceptible chickens by means of intratracheal swabs up to the time of this report is shown in table I.

Four of the chronic carriers (V-2270, ME-23086, V-2347 and V-2375) had laryngotracheal râles. The longest and most persistent carriers fell in this group. Definite areas of inflammation, some of which were covered with pseudomembrane, were found in the larynges and tracheas of these birds at necropsy. Photographs of lesions in the larynges of V-2375 and V-2347 are shown in figures 1 and 2.

Seventeen of the chronic carriers (V-2370, V-2290, V-2320, V-2321, V-2383, V-8225, V-1200, M-72012, V-1260, V-8277, V-1231, M-72010, J-3835, V-1269, M-4833, J-3393 and V-2118) did not have laryngotracheal râles, although the tracheas were not so moist as normal or in the acute stages of the disease, and histological examination revealed hyperplasia and loss of mucous glands and goblet-cells. V-2118 had papillae of pseudomembrane hanging on the outer margin of the larynx, as shown in

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TABLE I—*Data on periods during which chronic carriers eliminated virus.*

BIRD	VIRUS	VIRUS ELIMINATED (DAYS)
V-2270	Massachusetts	467
ME-23086	Massachusetts	467
V-2370	Massachusetts	252
V-2290	California	234
V-2320	Massachusetts	225
V-2321	New Jersey	211
V-2347	Massachusetts	205
V-2383	Massachusetts	200
V-8225	California	196
V-2375	Massachusetts	189
V-1200	Massachusetts	182
M-72012	Massachusetts	178
V-1260	Massachusetts	165
V-8277	New Jersey	154
V-1231	Massachusetts	140
M-72010	Massachusetts	127
J-3835	California	120
V-1269	Massachusetts	98
M-4833	Massachusetts	94
J-3393	Massachusetts	67
V-2097	Massachusetts	62
V-2118	Massachusetts	37

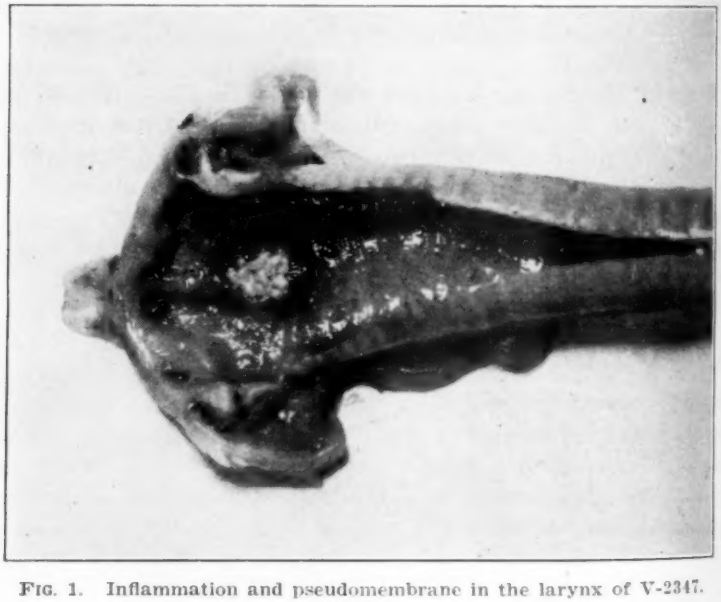

FIG. 1. Inflammation and pseudomembrane in the larynx of V-2347.

figure 3. In some of the tracheas the mucous glands were occluded, leading to the formation of small ulcers. The virus was found in the papillae of V-2118 and in either the larynx or trachea of the other birds not having laryngotracheal râles.

Whether a bird showed laryngotracheal râles or not seemed to depend upon the size of the areas of pseudomembrane obstructing the respiration and the extent of the inflammation in the larynx and trachea. The location of the lesions was important, for when they occurred in the larynx they were most likely to interfere with respiration and produce râles.

FIG. 2 (left). Inflammation and pseudomembrane in the rima glottis of V-2375.
FIG. 3 (right). Papillae of pseudomembrane on the outer edge of the larynx of V-2118.

Eight of the chronic carriers (V-2270, ME-23086, V-2347, V-2375, M-72012, M-72010, M-4833 and V-2118) were still eliminating virus at the time this experiment was closed, and the remaining thirteen had ceased to be carriers as far as could be determined by intratracheal swabs.

Four other birds recovering from infectious laryngotracheitis and showing persistent laryngotracheal râles were kept on the experiment the same as the chronic carriers, but they did not prove to be eliminating virus at any time. The most important data concerning these birds are shown in table II.

TABLE II—*Data on four recovered cases that did not eliminate virus.*

BIRD	NUMBER OF TESTS FOR VIRUS	DAYS ON EXPERIMENT
M-72011	9	124
P-120	7	98
V-2367	6	75
M-72015	5	65

The results of these observations and experiments on infectious laryngotracheitis are important because some poultrymen are endeavoring to identify carriers by intratracheal râles. Because of the small value of the individual, the fact that some birds having laryngotracheal râles are not carriers is not so important as the finding that most of the real offenders are likely to be missed because they do not show any evidence of laryngotracheal râles. As yet no reliable test is known for detecting carriers of infectious laryngotracheitis. Intratracheal swabbing of susceptible chickens is expensive, time-consuming, and not entirely reliable, because it is not always possible to touch the infected spot in the larynx or trachea of the living carrier. While this method has been used experimentally to determine the existence of chronic carriers and to learn something of their importance in transmitting the disease, it is not practical for field work.

SUMMARY

1. Chronic carriers of infectious laryngotracheitis have been found eliminating virus up to 467 days. At the end of this period, two fowls were still transmitting the disease to susceptible chickens at regular tests.

2. Four of the chronic carriers had laryngotracheal râles and seventeen did not show this symptom.

3. Four other birds recovering from infectious laryngotracheitis and having persistent laryngotracheal râles did not eliminate the virus as indicated by intratracheal swabbing into susceptible chickens.

4. In the 21 chronic carriers studied, the virus seemed to be confined to some portion of the larynx or trachea and lesions such as inflammation, hyperplasia, ulceration, and pseudomembrane appeared to be associated with it.

5. The results of these observations and experiments indicate that laryngotracheal râles are not a reliable means of detecting chronic carriers.

ACKNOWLEDGMENT

In conclusion, the writer wishes to extend his sincere thanks to Dr. Glen L. Dunlap, Department of Veterinary Science, Massachusetts Agricultural Experiment Station, for making the photographs accompanying this paper.

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OBSERVATIONS ON THE TOXICITY OF GOLDEN GLOW (RUDBECKIA LACINIATA) TO SWINE AND OTHER ANIMALS*

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INTRODUCTION

Rudbeckia laciniata, commonly known as golden glow, tall coneflower or thimble weed, belongs to the family of Compositæ. The Compositæ include about one-tenth of all the species of higher plants, and about one-eighth of all species in *Gray's Manual*. It is by far the largest family of flowering plants, although the number actually known to poison live stock is small. Some of the plants in this family are generally known to be poisonous to live stock, such as the cockleburs (species of Xanthium).[1, 2, 3] The white snakeroot (*Eupatorium urticæfolium*)[4, 5]; and two species of rayless goldenrod (*Aplopappus heterephyllus* and *Aplopappus fruticosus*),[6] affect animals and also cause trembles or milk-sickness in man. The sneezeweed (*Helenium autumnale*)[7, 1] is poisonous to horses, cattle and sheep. Some species of Senecio[8, 9] affect horses and cattle. The spring rabbit-bush (*Tetradymia glabrata*),[10] closely related to Senecio, has been found to kill sheep in Nevada. The ragweeds also belong to the Compositæ family and are notable for producing hay fever in man. The genus Rudbeckia comprises twenty-seven species.

DESCRIPTION OF GOLDEN GLOW

The following description of the plant, a perennial herb, is taken from Chestnut and Wilcox[11]:

> . . . attains a height of from 2 to 7 feet, and is usually smooth, except for a slight roughness of the margins and upper surface of the leaves. The root leaves are divided into from 5 to 7 parts and those of the lower portion of the stem into 3 to 5 parts, while the uppermost leaves are 3 parted. The flower heads are from 2 to 3 inches across, with yellow drooping rays and a dull yellow disk.

Golden glow (figs. 1 and 2) is also called wild sunflower in Montana. The well-known tall Rudbeckia grown for ornamental purposes has double, golden-yellow flowers.

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DISTRIBUTION

Golden glow is distributed from New Mexico to Manitoba and eastward across the continent. It is found in moist ground, along the banks of streams and in woods and thickets. In such places one is very likely to find species of water hemlock (Cicuta) also. The authors suspect that some cases of poisoning attributed to *R. laciniata* are due to other poisonous plants growing with it and not noticed, such as water hemlock, white snakeroot and others. Peterson[12] has reported the finding of *R. laciniata* in the

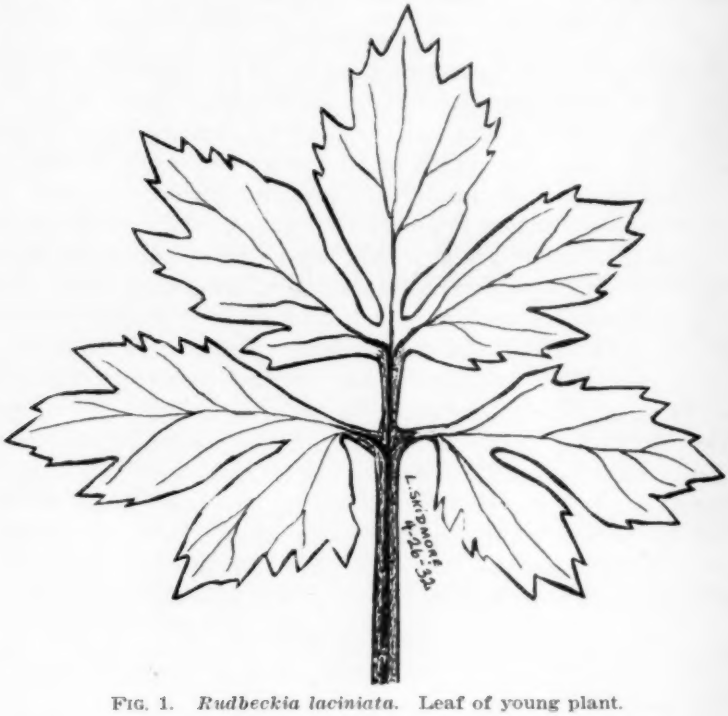

FIG. 1. *Rudbeckia laciniata.* Leaf of young plant.

southeastern part of Nebraska, also in the vicinity of Franklin, Lincoln, Nebraska City, Newark, Riverton, Saltillo, Wahoo and Wymore, all in Nebraska.

POISONOUS PLANTS OF THE GENUS RUDBECKIA

According to Pammel,[13] two species of Rudbeckia are reported to be poisonous to live stock, *viz.*, *R. laciniata*, discussed in this paper and said to be poisonous to sheep, and *R. occidentalis*, thought to have poisoned cattle in Kansas and Iowa. Chestnut and Wilcox[11] record an instance where 100 sheep were turned

into a two-acre timber-plot and fed mainly on *R. laciniata*. On the second day, 20 sheep showed symptoms of poisoning and 7 died. They state also that specimens of the plant have been sent from Missouri, with the complaint that it is not infrequently fatal to hogs. Gates[14] reports golden glow poisoning in swine. He cites a case:

> . . . a number of hogs, which had access to nothing else, died in convulsions with general symptoms of belladonna poisoning. . . . Fencing the animals from access to Rudbeckia prevented further trouble. However, the further feeding of Rudbeckia resulted in the death of the hog to which it was fed.

FIG. 2. *Rudbeckia laciniata* (after Britton and Brown).

Hansen[1] writes the following regarding black-eyed Susan (*Rudbeckia hirta*):

> Although experimental work on the subject is lacking, there is abundant field evidence pointing to this common wild and orna-

mental species as the cause of poisoning in cattle and hogs and a number of cases of this character have been observed in Indiana. In cattle, the trouble is characterized by severe digestive disturbance, colic, groaning, and passing of parts of the lining membrane in the feces. In hogs, loss of consciousness, followed by awakening and walking aimlessly for hours, has been observed as a result of grazing on the roots and tops of black-eyed Susan.

We have received occasional inquiries concerning the poisonous properties of golden glow to swine. We could find very little definite information in the literature on this subject, so we decided to answer the question by actual feeding trials.

SWINE

Experiment 1: On May 5, 1931, a quantity of *R. laciniata* plants, ranging in size from 3 to 8 inches tall, were gathered in the vicinity of Lincoln, Neb. There were also a number of water hemlock (Cicuta) plants in the area from which these plants were obtained. Two pigs weighing about 80 pounds each, which had been kept in a dry pen for some time, were fasted for 36 hours. They ate 4½ pounds of the whole plants, stems, leaves and roots. Each day from May 6 to 10, inclusive, they ate 3 pounds of the ground-up plants (meat-grinder used), stems, leaves and roots, which were mixed with a little ground feed consisting of oats and corn. This was all the feed these pigs received during this time. They ate all that was given to them each day and were allowed a liberal amount of water daily. During this feeding trial, these pigs always appeared normal and at no time did they show any symptoms of poisoning. The two pigs consumed a total of 19½ pounds of *R. laciniata* without showing symptoms of poisoning.

The plant at this stage of growth did not prove toxic to swine, even though it is usually held under suspicion in early spring, when green vegetation is scarce and when the plants are quite small.

Experiment 2: On July 10, 1931, another feeding trial was conducted with more mature plants, which were 2 to 3 feet tall and had new shoots on stooling roots. Four hogs (approximate weight, 125 pounds each, were put into a pen with a concrete floor and fasted for 40 hours. A liberal supply of water was allowed. These animals were given 42 pounds of whole plants, of which they consumed about 16 pounds. Within 24 hours, all hogs showed incoördination in walking. Respirations were greatly accelerated, ranging from 90 to 114 per minute. The curl was lost from the tail, which hung down straight. Tempera-

tures in all remained normal. Two animals showed abdominal pain, evidenced by stretching their bodies while in a standing position. They refused all feed and drank but very little water. There was slight dribbling of saliva from the mouth and occasional champing of the jaws. For almost 36 hours, one hog walked aimlessly from one end of the pen to the other. This animal was not seen to lie down during this time. Three of the hogs began to eat a little within 24 hours. The severely affected animal did not eat for 36 hours. The daily feeding of 4 to 6 pounds of ground-up Rudbeckia, mixed with an equal amount of feed consisting of finely ground oats, wheat, corn and tankage, was continued until July 25, at which time the animals were discharged from the experiment, in apparently normal condition, with the exception of considerable loss in weight. These animals consumed a total of 57 pounds of *R. laciniata* without fatal results. The first feeding of plants, after a fast, produced symptoms. The swine then apparently developed a tolerance for the plant although they never relished eating it, as golden glow has a very disagreeable odor and taste.

Experiment 3: On July 15, 1931, three pigs weighing 55, 60 and 65 pounds, respectively, which had been fasted 48 hours, were fed 35 pounds of the whole plants. Within 24 hours they ate seven pounds. At the end of this time, the three animals showed incoördination of hind legs in locomotion, marked increase in respiration, ranging from 90 to 104 per minute. The temperatures remained normal. There was slight dribbling of saliva from the mouth and occasional champing of the jaws. One pig had abdominal pains. At the end of the next 24 hours, two pigs appeared almost normal. However, pulmonic disturbance was still present in all. One pig was still very nervous. It would not lie down but walked aimlessly for 36 hours, from one end of the pen to the other, with marked incoördination of the hind legs. These three animals consumed a total of 24 pounds of *R. laciniata.* On July 26, feeding of the plants was discontinued. The pigs appeared normal, but had lost considerable weight.

SHEEP

Experiment 1: July 17, 1931: Two sheep were put into a small pen, from which all bedding was removed, and were fasted for 48 hours. They were then fed 25 pounds of *R. laciniata,* 6 pounds of which they ate in 24 hours. Then both sheep showed marked increases in respiration, although temperatures were normal. The animals were droopy and listless, were unsteady

on their legs, with incoördination of movements. One sheep kept its neck stretched, with the nose raised upward, making up and down movements with the head. On July 18, they were fed 3 pounds of ground-up plants mixed with 5 pounds of oats, which they refused to eat. The next day, one animal appeared almost normal. The other was still in distress. They were fed 3 pounds of fresh plants each day until July 25, at which time the feeding was stopped. Both animals appeared normal, with the exception of marked weakness brought on by starvation, because they would not eat the plants.

RABBITS

Experiment 1: July 20, 1931: Ten rabbits fasted for 60 hours were fed 15 pounds of *R. laciniata*. Each day two pounds of fresh plants were fed. On July 22, one rabbit had partial paralysis of the hind legs. This animal died the following day. On July 26, another rabbit had paralysis of the hind legs and died. Bacteriologic findings in both animals were negative. Both showed marked pulmonary disturbance. Microscopic examination of the internal organs showed normal kidneys and spleen, both showed pneumonia and one rabbit had fatty degeneration of the liver. Feeding was discontinued July 26, at which time the remaining animals were in a moribund condition.

Experiment 2: July 25, 1931: Ten rabbits were fasted for 48 hours and then fed 15 pounds of whole plants. Each day they received 2 pounds of fresh Rudbeckia. July 29, one rabbit died. July 30, another rabbit died. Both animals showed marked pulmonary disturbance, and paralysis of the hind parts. Bacteriologic findings were negative. On autopsy both had slightly enlarged livers, with slight brownish-yellow mottling. The kidneys showed slight congestion. There was marked evidence of pneumonia in both rabbits. July 31, feeding was discontinued, as the animals were rapidly losing weight.

CAVIES

Experiment 1: July 20, 1931: Ten cavies fasted for 60 hours were fed 10 pounds of *R. laciniata*. They received 2 pounds of fresh plants each day. July 24, one cavy died. Autopsy findings: liver, slight congestion, brownish-yellow mottling; stomach, well filled, slight congestion and mild gastritis; intestines, normal; lungs showed pneumonia. July 26, another cavy died, with paralysis of the hind legs. Autopsy findings were the same as described previously. Bacteriologic findings were negative in both

animals. The cavies ate the plants fairly well the first two days; after this they ate very little and became quite thin. Feeding was discontinued, July 26.

Experiment 2: July 25, 1931: Ten cavies were fasted for 48 hours and then fed 10 pounds of *R. laciniata.* They were fed 2 pounds of fresh plants each day. Within one week, three animals died. Autopsy revealed the same lesions as described in the previous experiment. Bacteriological findings were likewise negative. A microscopic examination was made of three of the cavies which died in these feeding trials. All three showed fatty degeneration of the liver, and two had pneumonia. The kidneys and spleen in two cavies were apparently normal, while one showed a slight nephritis and siderosis of the spleen. When the feeding was discontinued, these animals were very thin.

SUMMARY

Rudbeckia laciniata, commonly known as golden glow, is a widely distributed plant, growing along the banks of streams and in moist places. In such places one is also very likely to find species of hemlock and white snakeroot, which are known to be poisonous to live stock. A few cases of *R. laciniata* poisoning in domestic animals have been reported. Our feeding trials were carried out to give more information on the toxicity of this plant to swine, sheep, cavies and rabbits. Two pigs, weighing 80 pounds each, consumed a total of 19½ pounds of young, whole plants of *R. laciniata* without showing any symptoms of poisoning. The more mature plants, when fed to seven other hogs, produced toxic conditions in swine characterized by loss of appetite, nervous symptoms, incoördination of movements, increased respiration and aimless roaming about, with apparent visual disturbance. These symptoms lasted from 24 to 36 hours. Then the appetite returned and continued feeding failed to produce a fatality in any of the swine. They appeared to develop a tolerance for the plant. No postmortems of swine were made to determine if any damage was done to the body organs. In sheep, likewise, similar symptoms were produced, but the animals did not die from eating golden glow. Sheep will not eat enough of the plants to sustain life.

The feeding of *R. laciniata* to rabbits and guinea pigs did kill some of these animals. They ate so little of the golden glow that they would starve to death rather than eat this disagreeable tasting and smelling plant. Animals which have been deprived of food and are hungry will eat golden glow in sufficient quan-

tity to produce toxic effects. After the first feeding, it is almost impossible to get animals to eat the plant unless they are forced to do so and even then they will starve to death because they will not eat enough to sustain life. *R. laciniata* is more toxic in the mature state of growth. Microscopic examination of the organs of four of the five rabbits and cavies which died showed fatty degeneration of the liver and pneumonia.

Where good feeding practices are followed, there should be practically no danger from *R. laciniata*, because the plants have a very disagreeable taste and will not be eaten by live stock unless they are very hungry or poorly fed. Care should be exercised also in turning live stock into wooded pastures, low marshes, and along streams in the spring, before the desirable vegetation has a good start. Likewise, there is always danger in overgrazing an area, particularly during the dry part of the season. Pastures, sometimes in early and late fall, may have very little left for animals to eat except dangerous plants.

ACKNOWLEDGMENTS

The authors express their appreciation for the coöperation given by the Department of Animal Husbandry; Dr. S. W. Alford, of the Nebraska Agricultural Experiment Station; Dr. K. Wagener, Berlin Veterinary School, Germany, who assisted in gathering plants for most of these feeding experiments, and S. Fred Prince, who drew figure 2 (after Britton and Brown).

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JOHNSON GRASS (SORGHUM HALEPENSE) POISONING*

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Loco Weed Laboratory, Texas Agricultural Experiment Station†

In the Davis Mountains region of western Texas, Johnson grass is frequently planted in flats which are flooded from the surrounding hills. In favorable seasons these flats are a valuable source of forage for winter feeding. However, the grass is frequently the cause of important cattle losses, owing to the fact that the potential poisonous property of the plant is not appreciated and the fields are pastured quite extensively during periods of drouth. Furthermore, the fields are the source of seed for small stands of volunteer grass which escape the boundary fences and are available to range animals at all times. That the losses sustained from these sources are frequently quite extensive, in Brewster and Presidio counties, is shown by the following reports:

Case 1: On the afternoon of May 17, 1930, five head of dairy cattle were being herded on a flat, which, in addition to other forage, contained a scattered stand of volunteer Johnson grass. Three of the cows began to show alarming symptoms within fifteen minutes from the time they started to eat the grass, and the herder drove the animals from that particular area. Two of the cows died within ten minutes from the time the first symptoms appeared. The third animal recovered. The two remaining animals showed no evidence of poisoning.

Case 2: During the afternoon of May 24, 1930, 50 head of dairy cattle were being herded on a flat adjacent to the flat which figured in the preceding outbreak. The forage conditions associated with the two outbreaks were identical. Within thirty minutes from the time the first animals arrived in the area containing the Johnson grass, two of them began to show symptoms

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†In coöperation with the Bureau of Animal Industry, U. S. Department of Agriculture.

of excitement and distress, and the herder started to drive the herd to the corral. During the next ten minutes four head "went down," three of which died almost immediately, but the fourth eventually recovered. The remainder of the herd showed no evidence of poisoning.

Case 3: On the morning of June 1, 1930, 120 head of cattle were driven across a narrow field of Johnson grass. The stand of grass was very scattered and, at the point crossed, the field was not over 300 yards in width. Three of the animals showed symptoms of poisoning and died within ten minutes from the time they left the area containing the grass. The remainder of the herd was not affected.

Case 4: Some time before dark, on the night of June 9, 1930, 100 head of yearling heifers were turned into a small pasture. Within this pasture, and not isolated by a boundary fence, was located the Johnson-grass field which was responsible for the losses in the preceding outbreak. The cattle were not observed from the time they were turned out until about sun-up the next day, at which time ten head were found dead in the Johnson-grass area. Nothing was known as to the number which may have grazed on the grass, as the remainder of the herd was some distance from the field and none of them showed evidence of poisoning.

Case 5: On the morning of May 24, 1932, 85 head of cattle were being driven through a lane. A small amount of volunteer Johnson grass was growing along the road for a distance of about 100 yards. As the animals reached the grass they were permitted to graze. Symptoms of poisoning appeared in two of the leaders within five minutes from the time they began to eat the grass. During the next five minutes, several more became affected but they were not immediately noticed as the owner's attention was concentrated on the actions of the first two. When the seriousness of the situation became apparent the animals were driven from the area as rapidly as possible, but by this time 24 head had shown symptoms of poisoning. Sixteen of the 24 died during the next few minutes. The remainder survived the effects of the poison. However, the chances for an ultimate recovery were rendered somewhat doubtful as the owner performed rumenotomies on these cases.

Case 6: On the morning of June 21, 1932, a Jersey cow was permitted to graze on a Johnson-grass field. The animal was alive and apparently well at noon, but was found dead about two hours later. A range animal was found dead about the

same hour a short distance from this field of grass. Nearby this animal was a small patch of volunteer Johnson grass, the bitten tops of which suggested that about a pound had been eaten. Since the tracks in the soft dirt in the vicinity of the carcasses failed to suggest either struggle or agony, it was assumed that death was rather sudden in both cases.

Case 7: On the afternoon of June 21, 1932, 225 head of yearling cattle were turned into an old Johnson-grass field in which most of the grass had died out. Two hours later, two of the animals were found dead beside a small patch of the grass. This herd was removed, but two or three days later another herd of 75 animals was placed in the same field in order to hold them for a short time. Some time within the next two hours, two animals were found dead near the spot where the other two had died. In both cases some of the remaining animals had eaten some of the grass but evidently not in quantities sufficient to produce toxic effects.

In addition to the cases reported, a number of cases in which but a single animal was affected have been observed. In these cases the animals generally gained access to the grass by forcing their way through protecting fences.

There was no opportunity to make personal observations on the symptoms of acute, fatal poisoning, but they were reported to consist of the sudden appearance of symptoms of excitement and distress, such as running backwards, bawling, shaking of the head, opisthotonos and frantic efforts to remain standing. The initial symptoms were soon followed by paralysis, stupor and death. Sublethal doses were manifested by the initial symptoms of excitement and distress, but milder in appearance. These symptoms were generally followed by a period of depression, during which time the animals remained prostrate and in a semicomatose condition for variable periods of time. As the toxic effects wore off, the animals would get up and wander about in a dazed condition, with pronounced muscular incoördination, especially noticeable in the rear quarters. In this condition the animal exhibited an attitude similar to one shown by an animal partially recovered from a stunning blow on the head or from a general anesthetic. Complete recovery was generally uneventful and required from 24 to 36 hours.

The occurrence of these cases has so far been confined to the late spring and early summer months. The fact that no cases have been associated with frosted Johnson grass lends some support to the opinion of some ranchers to the effect that Johnson

grass is not poisonous during the fall and winter months. To assume from the present observations that this is a settled fact would be unwarranted. However, it is evident that frosting is not so important a factor in this type of poisoning in the Big Bend area as it is further north, where poisoning by the sorghums is frequently associated with a frosted condition of the plant.

The climatic conditions and the condition of the grass have been quite constant in all the outbreaks. A period of drouth, varying from days to weeks, has preceded every outbreak. At the time the losses occurred, the growth of the grass had been arrested in some cases to the extent of sunburnt tips; in others, nothing further than noticeable wilting had occurred. The fatalities occurred on grass three to six inches high, which had not reached the jointed stage. However, in some places the older jointed grass was present but had not been bitten off to a noticeable extent. Therefore, a preference for the younger, tender blades of grass may explain the constant association of this factor with the fatalities rather than a non-toxic condition of the older plants.

AN OUTBREAK OF TRICHINOSIS IN PORTLAND, OREGON*

By E. E. CHASE, *Portland, Ore.*

Chief Meat Inspector, Bureau of Health

Several cases of trichinosis recently occurred in Portland, following the eating of salami, or summer sausage, which was prepared in one of the local plants. This establishment is under municipal inspection.

Meat inspection in Portland dates back to March, 1916, at which time the writer was appointed Chief Meat Inspector. The ordinance passed at that time was a very good one, with but few exceptions. For example, regulations were not provided for the preparation of meat food products containing pork muscle tissue to be eaten without being cooked. An amendment to the ordinance was prepared at the time and submitted to the Council, but it was defeated at the request of the sausage-makers.

About August 25, 1932, a family living in Portland gave a dinner party to which seven guests were invited. Salami, or summer sausage, was served at the dinner, and was eaten by

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all those present. About ten days later, these persons began to notice the following symptoms: swollen eyelids (almost closing the eyes for a period of two days), face somewhat swollen, temperature ranging from 100° F. in the morning to 105° F. at night. In one case, the patient seemed to have double vision, and some of the others complained of pains in the back and loins. All complained of dizziness and weakness. Three of the persons had profuse diarrhea.

On September 7, Dr. Herbert Nichols reported to our office that the hostess of the dinner party mentioned in the preceding paragraph was at the Good Samaritan Hospital, and that her case had been diagnosed as trichinosis. The writer understands that Dr. Nichols had had her under his care for several days, suspecting typhoid fever. The diagnosis of trichinosis was made by Dr. Manlove, bacteriologist at the hospital, on the findings of a blood count. As soon as the report came to our office, we immediately informed the press.

On the evening of September 7, three more cases of trichinosis were reported by Dr. Tracy Parker. The symptoms in these cases were about the same as in the others. On the following day, Dr. Uhle reported three other cases, all in one family. The writer immediately called on the family and interviewed the mother. Her three sons were all affected with trichinosis, and had about the same symptoms as the others, except that diarrhea was absent, but there was more or less pain in the legs. In the case of the 12-year-old son, there was complaint of pains in the chest, in the region of the diaphragm. As soon as Dr. Uhle saw the accounts in the press, which gave the symptoms of trichinosis, he took a sample of blood from the boy and sent it to the Emanuel Hospital. The diagnosis was trichinosis. Dr. Uhle at first had suspected typhoid.

On September 9, Dr. D. C. Bassett, assistant meat inspector for the City of Portland, who had been on his vacation, informed the writer that he had been ill for several days and under the care of Dr. Stewart, of Oregon City. His symptoms, too, were suggestive of typhoid fever. Dr. Louis Wolf, assistant health officer for the City of Portland, and the writer visited Dr. Bassett, in consultation with Dr. Stewart. He then had been sick for about a week. At the suggestion of Dr. Wolf, five cubic centimeters of blood was drawn from his arm and submitted to the city laboratory. One larva was found in the blood, and a diagnosis of trichinosis was made. Diarrhea was absent in Dr. Bassett's case. He complained, however, of feeling weak.

It was not possible to find any of the sausage which caused the trouble. In every case all of it had been consumed. It was manufactured by a local sausage-plant. Dr. Bassett was the inspector at this plant and had purchased salami there on several occasions. Every case of trichinosis was traced to sausage from this plant.

In the family where the three boys were affected, four others ate of the same sausage and were not affected. Dr. Bassett's wife appears to be all right. Probably the infestation was a slight one, because the patients did not complain of severe muscular pains. To date, no more cases have been reported.

On September 22, an ordinance was presented to the Council, which provides that no articles of food consisting wholly or in part of pork muscle tissue, and which are of a kind customarily prepared in meat-handling establishments to be eaten by consumers without being cooked, shall be kept, offered or exposed for sale as food for human consumption, unless the pork muscle tissue entering into the product shall have been subjected to heat, either before or after its inclusion in the finished product, so that all portions of the pork muscle tissue shall have attained a temperature not lower than 137° F., or, as an alternative for this heating method, unless the article of which the pork muscle tissue is an ingredient shall have been subjected to refrigeration for a continuous period of not less than twenty days at a temperature not higher than 5° F.

This ordinance was passed as an emergency measure on September 22, and is now being enforced. The salami which caused this outbreak was neither brought to a heating point of 137° F., nor was it refrigerated sufficiently to kill the parasite. The sausage-makers are all willing to coöperate with the Bureau of Health under the new ordinance.

A RICKETTSIA-LIKE OR ANAPLASMOSIS-LIKE DISEASE IN SWINE*

By L. P. DOYLE, *Lafayette, Indiana*

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During the past few years, cases have been encountered in swine which show fairly well-defined clinical features, but which are more particularly characterized by the distinctive pathologic

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features revealed on postmortem examination. Apparently this disease has not been described previously. Thus far, the writer has encountered the disease only in shotes from two or three months to six or eight months of age.

While the mortality in visibly affected animals has been high, the death rate in herds has not been observed to exceed ten per cent. Usually not more than one to three per cent of the herd die from this disease. One instance was observed where 12 shotes in a herd of 200 died. This herd had been assembled from several sources. The owner stated that he believed all the deaths occurred in a group of 30 pigs which had been purchased from a neighboring farm two months previously.

The clinical and postmortem features of the disease may well be characterized as an ictero-anemia. The clinical course of the observed cases has been quite rapid—lasting from one and one-half to four or five days. Ordinarily, the first symptom noted is depression; later there is weakness and usually dyspnea; and finally icterus and anemia frequently become quite apparent in the mucous membrane of the mouth and in the skin of animals which do not have much natural pigment. According to some hog-owners, the affected animals frequently show evidence of "great pain" a short time before they die. Elevated body temperature (104 to 105° F.) have been noted in several cases.

The postmortem findings are very characteristic, or even pathognomonic. In animals which are moribund, the blood is very pale; the plasma or the serum is yellowish, and the red cells sometimes show spontaneous agglutination. The blood usually clots fairly readily after it escapes from the body. Icterus is a striking feature of the postmortem findings. The spleen is greatly enlarged and softer than normal. The liver shows yellowish discoloration; and the heart muscle and the kidney parenchyma show gross evidence of degenerative changes. Ascites, hydrothorax and hydropericardium, characterized by the presence of yellowish (icteric) fluid, are sometimes found. The contents of the intestine, and even of the stomach, frequently show marked yellowish coloration due to bile pigments.

Microscopic examination of the blood showed a well-marked increase in the number of neutrophilic polymorphs; irregularity in the size and shape of the red cells; and occasionally a macrophagic cell containing one or more red cells. Normoblasts were not numerous. Blood smears stained with Giemsa's stain showed the presence of bodies, evidently organismal in nature, within the red cells. These bodies showed considerable variation in

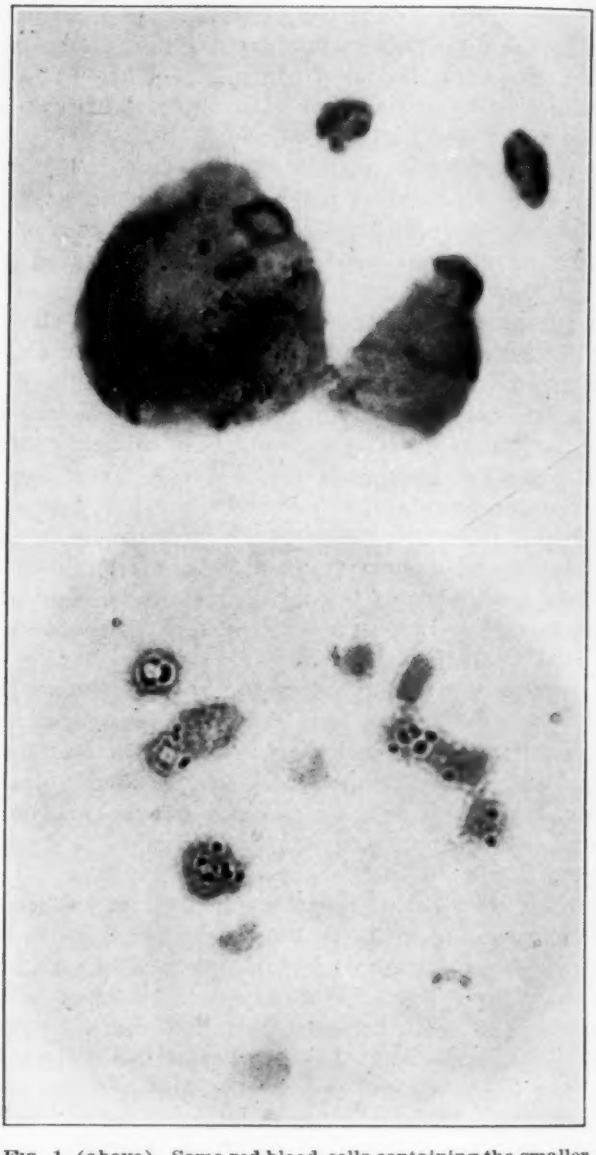

FIG. 1 (above). Some red blood-cells containing the smaller cell inclusions. These cell inclusions are coccoid, bacilliform and ring-form. The affected red cells are swollen somewhat, but their apparent great size is due largely to clumping or fusion of two or more red cells.

FIG. 2 (below). Some red cells containing the larger cell inclusions. Each cell inclusion is surrounded by a clear zone.

morphology. Some were coccoid, some bacilliform, while still others were ring-formed. The ring-formed bodies often showed small chromatin knots or masses at certain points on their circumference. Some of the erythrocytes were almost filled with these bodies; and many of the bodies were free from the red cells. The free bodies very likely came from disintegrated erythrocytes, since some of these cells were seen to be undergoing disintegration.

In some instances, following intense staining in Giemsa's stain, much larger inclusions than those described above were found within the red cells. These larger inclusions were fewer in number than the small ones. They varied considerably in shape and were usually surrounded by what appeared to be clear zones.

Discussion

The disease reported here has some of the clinical and gross pathologic features of anaplasmosis. The type of cell inclusion commonly found, however, is perhaps more suggestive of a rickettsia than anaplasmosis. The fact that ordinarily only a small portion of a herd is visibly affected by the disease suggests that either some limited agent spreads the causative factor or some special condition is required for the disease to develop when the specific factor is present.

AZAMINE IN THE CONTROL OF BOVINE COCCIDIOSIS*

By Charles R. Eno, *Red Hook, N. Y.*

Though known to veterinarians for many years—since its demonstration as a clinical entity by Zschokke, in 1892—bovine coccidiosis has until recently been classed as a rare disease. No scientific interest was apparently taken in the causal organism, at least in the United States, before Crawley[1] described it in his contribution to the annual report of the Bureau of Animal Industry for 1910. In the quarter-century which has elapsed since his paper was written, the American veterinary profession has come to recognize coccidiosis as among the serious menaces to the health of cattle, but this lapse of time has seen no noticeable advance in either its diagnosis or treatment.

The disease, commonly known as "red diarrhea," is due to the presence in the intestine of the affected animal of the protozoan organism, *Eimeria* (*Coccidium*) *zürni*, which attacks the

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cells of the epithelial lining of the lower bowel. It is essentially a disease of young stock, and when adult animals fail to develop the characteristic manifestations after exposure to the infection, it is probable that they have suffered an attack during calfhood, the evidences of which were so slight that they were overlooked by those in charge of the animals.

Blood in the feces is the only pathognomonic sign, and as this may be due to a variety of causes other than the coccidium, the only positive diagnosis is that made by laboratory examination of fecal specimens and study of smears and portions of epithelium scraped from the rectum near the anus. In these the causal organism can be detected readily.

The coccidium appears in the bowel discharge in the oöcyst stage and, when absent from the feces, may sometimes be found in the epithelial scrapings, hence the importance of obtaining this material if there is any doubt about the nature of the infection. The mucous membrane of the bowel wall will be noticeably pale in color, due to the loss of blood following cell-destruction by the activities of the parasites.

The fact that a wide variety of remedial measures have been offered, from the first recognition of the disease, indicates that nothing has yet been found that is entirely satisfactory. Of course, the usual precautions against infection spreading to unaffected animals must be taken. The sick ones should be isolated, and the greatest care used in disposing of the excreta. While proper disinfection is requisite, it should be kept in mind that no disinfectants will serve *in place of* cleanliness. The tremendous loss of blood and fluid from the bowel must be counteracted by proper supportive measures, and the animal made as comfortable as possible in every way.

The writer has used various preparations in treating this disease, but without brilliant results in the past. In a recent outbreak of coccidiosis in a fairly large herd of adult animals, he made use of a new preparation, azamine, which gave such good results, both in the elimination of the infection in those animals which were in an advanced stage of the disease when first seen, as well as in protecting those still unaffected from contracting it, that it is deemed worth while to report these results here.

The herd concerned consisted of a large number of Aberdeen Angus steers, and a number of cows of the dairy breeds. They were kept under sanitary conditions and handled with intelligence above the average. When first seen by the writer, some twenty-five steers already displayed undoubted clinical signs of

the infection. A number of cows in the dairy herd, one of them more than eight years of age, were also in the acute stage.

The diagnosis was confirmed by microscopic examination of the bowel discharges from several different affected animals, and also by inspection of tissue scrapings from the rectum in certain cases. These examinations were made by the laboratory of the New York State Veterinary College, which reported: "Positive for *Coccidium (Eimeria) zürni.*"

Medication consisted in immediate institution of the general supportive measures already mentioned and the administration of 5-gram capsules of azamine, by mouth, three times daily. In addition to this treatment of active cases, 50 steers were given azamine in powdered form as a preventive. The powder was removed from the capsules and sprinkled over their feed. Not one of these steers showed the slightest evidence of "red diarrhea" at any time, all remaining in perfectly good condition for the duration of the outbreak.

Only one of the actively infected steers died, and there was no loss in the dairy herd, although the 8-year-old cow was extremely ill, so that the writer quite despaired of her recovery. The unit of dosage employed was a 5-gram "horse" capsule. These were administered two or three times each day, according to the age of the animal and the virulence of the infection.

The experience in this series of cases leads the writer to believe that in azamine the veterinary profession has obtained a very useful agent for combating what has heretofore been a condition peculiarly hard to control and eradicate. It is certainly deserving of extensive clinical trial.

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Dog Population in England

Canine population statistics are reported by a London newspaper with the observation that dogs are playing an increasingly important part in the lives of humans. Between 1831 and 1931, the human population of England and Wales increased nearly three times, while the canine population increased tenfold —from 300,000 to 3,000,000. During the past twenty years, human population has increased 11 per cent, while the number of dogs has doubled.

THE CHILD AND THE TUBERCULOSIS PROBLEM. J. Arthur Myers, Ph. D., M. D., F. A. C. P., Professor of Preventive Medicine, University of Minnesota; Chief of Medical Staff, Lymanhurst School for Tuberculous Children, Minneapolis, Minn. 230 pages, with 21 illustrations. Charles C. Thomas, Springfield, Ill., and Baltimore, Md., 1932. Cloth, $3.00 postpaid.

The latest information on reliable methods of discovering, controlling and preventing tuberculosis among children has been collected by the author and is presented in a very practical way. Much progress in our understanding of tuberculosis of childhood has been made in recent years and no person is better qualified to evaluate this new knowledge than Doctor Myers, a prominent member of the medical profession and a mighty good friend of the veterinary profession. Considerable evidence of the latter is found in the book.

The author looks upon tuberculosis in much the same way that we have come to look upon some of the acute infectious diseases of both man and animals. If we are to control or prevent these infections, we must put into practice our knowledge of their epidemiology. In the case of tuberculosis, as in typhoid, infected individuals must be located and isolated, in order to reduce to a minimum the possibility of communicating the disease to others. If isolation is impractical, the person may be taught how to prevent the spread of tubercle bacilli to others. The child's best chance for recovery is promoted when further exposure is stopped, according to Doctor Myers.

The book is divided into 16 chapters and two of these are of particular interest to the veterinarian, as they deal with the methods now in vogue in America for controlling tuberculosis among domestic animals, as well as other phases of the tuberculosis problem in which the physician and veterinarian have interests in common.

Doctor Myers acknowledges that as recently as 1927 he held

different views from those he now entertains on the subject of disposing of cattle reacting to the tuberculin test. At that time he questioned the advisability of slaughtering all known infected animals. In a splendid exhibition of magnanimity, he apologizes to the veterinarian for having placed obstacles in his way, and then admits that time has proved that the vision of the veterinarian was clearer than his own. Doctor Myers owes no apology to the veterinary profession. Time and again he has demonstrated by his actions that he is "with" the veterinarian.

As further evidence of just where the profession stands in medical circles, a paragraph is quoted from the book:

> SOME OF THE CONTRIBUTIONS OF THE VETERINARIAN.—I only wish that words could express the great appreciation that many members of the medical profession have of the remarkable accomplishments of the veterinarians and closely allied groups. Every time we see a man or a woman with a hunchback, we should pay tribute to the veterinarians, knowing that the possibilities for such deformities among the girls and boys of the present and the future have been greatly reduced by this group of professional workers.

Doctor Myers is to be congratulated on his latest literary effort. Veterinarians may well feel proud of the tributes paid the profession by the author of this book. It deserves a place on the bookshelf of every veterinarian.

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Kenya Colony and Protectorate Dept. of Agr. Annual Report, 1931. (Nairobi, Kenya Colony, E. Africa, 1932. pp. 350.)

Kenya Colony and Protectorate Agr. Census, 1932. (Dept. of Agr., Statistics Branch, Nairobi, Kenya Colony, E. Africa, 1932. pp. 56.)

A Preliminary Report on Investigations on the Buffalo Fly (*Lyperosia exigua* de Meij.) and Its Parasites in Java and Northern Australia. E. Handschin. (Pamph. 31, Coun. for Sci. & Ind. Res., Commonwealth of Australia. Melbourne, Australia, 1932. pp. 24.)

Report of the Nevada State Board Stock Commissioners for the Period January 1, 1931, to June 30, 1932. (Carson City, Nev., 1932. pp. 21.)

Report of the Nevada State Rabies Commission, for the Period January 1, 1931, to June 30, 1932. (Carson City, Nev., 1932. pp. 8.)

GIARDIA IN SHEEP IN VICTORIA, AUSTRALIA. A. W. Turner and D. MURNANE. Austral. Jour. Exp. Biol. & Med. Sci., x (1932), 1, p. 53.

The authors describe a case of heavy infestation with *Giardia* sp. coëxistent with the presence of enormous numbers of large Gram-positive organisms in the small intestines of a sheep. Emaciation over a period of several months, dullness, weakness, loss of appetite and marked paleness of the visible mucous membranes were among the symptoms. Autopsy revealed general bloodlessness of the body, watery and subnormal volume of blood, lungs extremely pale, and the small intestine contained 350 cc of fluid contents which contained 150 millions of the Gram-positive organisms per cubic centimeter and about one million free flagellates to the cubic centimeter. *Giardia* have a wide variety of hosts and in such abundant numbers are considered pathogenic.

CERTAIN CHEMICAL AND MORPHOLOGIC PHASES OF THE BLOOD OF NORMAL AND CHOLERA-INFECTED SWINE. I. The Concentration of certain chemical constituents. E. A. Hewitt. Iowa State Coll. Jour. of Sci. vi (1932), 2, p. 143.

The concentration of total non-protein nitrogen in the normal blood of swine was found to have a somewhat wider variation than that recorded in other species, although the average value of 31.4 milligrams of total non-protein nitrogen per 100 cc of blood agrees closely with the average value reported for other species. In some cases of hog cholera, the total non-protein nitrogen may increase to a very high level, while in other and perhaps most cases of hog cholera, the total non-protein nitrogen concentration of the blood is not disturbed. Lower values were found for urea nitrogen in normal swine blood than recorded by other investigators. Withholding food and water before slaughter may be a cause for this. Cholera-infected pigs showed still lower values for urea nitrogen and may represent a pro-

longation of starvation metabolism. Only limited evidence was obtained that would indicate renal impairment in hog cholera. The few instances of high total non-protein nitrogen values in the cholera-infected blood would indicate that there were nitrogen retentions in some cases. The slightly higher average value for creatinine in cholera-infected blood might suggest some renal impairment.

THE USE OF IODIN AND OF CERTAIN IODIN COMPOUNDS IN EXPERIMENTAL TUBERCULOSIS. Georges Knaysi. Jour. Inf. Dis., 1 (1932), 3, p. 261.

Iodin may be injected intravenously into the body of the rabbit over relatively long periods of time without apparent harm to the health of the animal or injury to the thyroid gland. The dose used in most of the experiments was two milligrams per kilogram body weight, administered twice a week over a period of about two months from an aqueous solution containing three times as much potassium iodid. Such a dose is not effective in the treatment for tuberculosis, probably because it is quickly neutralized by the alkali reserve of the blood. A dose of 30 mg. of iodin per kilo of body weight does not kill a normal animal, but one of 20 mg. per kilo of body weight is fatal to a rabbit with a severe miliary tuberculosis.

METABOLISM STUDIES ON THE BRUCELLA GROUP. III. Viability in aqueous solutions. C. E. ZoBell and Margaret H. ZoBell. Jour. Inf. Dis., 1 (1932), 5-6, p. 538.

In general, under practically all conditions that were tried, suis organisms were most resistant, melitensis intermediate in resistance, and abortus least resistant, as indicated by their survival times when suspended in aqueous solutions. In a balanced cystine salt solution, Brucella organisms in dilute suspensions remain viable for from four to five weeks at room temperature. This extended viability without clumping causes the authors to recommend it as a suspension fluid for standardized inocula.

AGGLUTININ-ABSORPTION STUDIES ON BRUCELLA. Wayne N. Plastridge and James G. McAlpine. Jour. Inf. Dis., 1 (1932), 5-6, p. 555.

The authors present agglutinin-absorption data obtained on 142 strains of Brucella of human, bovine, porcine, caprine and

equine origin. A comparison of the identity of these strains, as determined by their ability to utilize dextrose and their behavior on Huddleson's dye plates, with their serologic characteristics, shows that the agglutinin-absorption test failed to differentiate between *Br. abortus* and *Br. melitensis* as far as 23 (15 per cent) of the strains studied were concerned. This work confirms the work of Burnet, that while it is possible to classify some strains of *Br. melitensis* as such by means of the agglutinin-absorption test, it is impossible to identify strains of *Br. abortus* by means of this test with any degree of certainty. Evidence of variations in the serologic properties of the members of the genus Brucella, especially *Br. melitensis*, under ordinary laboratory conditions, are noted and discussed.

FACTORS INFLUENCING THE BLOOD-SUGAR LEVEL OF DAIRY CATTLE. R. E. Hodgson, W. H. Riddell and J. S. Hughes. Jour. Agr. Res., xliv (1932), 4, p. 357.

Blood samples from 140 dairy cattle were analyzed for sugar content. Calves shortly after birth had a sugar content of about 100 mg. per 100 cc. As the age of the animals increased, the blood sugar decreased, until the animal was approximately two years old, after which little further change was observed. A mean blood-sugar content of 53.03 ± 0.297 mgs. per 100 cc of blood was obtained on 222 samples from 74 animals between two and eight years of age, with a range of 35 to 74 mgs. No significant difference was observed in the four breeds studied. Cows giving a liberal flow of milk were found to have slightly less blood sugar than dry cows or those yielding a small quantity of milk. There was no increase after feeding; fasting caused a decrease in blood-sugar content of dairy heifers amounting approximately to 50 per cent. The administration of glucose in solution produced increases in the blood-sugar content, amounting to as much as 200 per cent. Excitement produced a marked increase in blood sugar. The blood-sugar values of cows and heifers were higher during oestrus than at other times.

STUDIES ON BOVINE MASTITIS. V. The more acute forms of streptococcus mastitis. F. C. Minett, A. W. Stableforth and S. J. Edwards. Jour. Comp. Path. & Therap., xlv (1932), p. 1.

The authors discuss a form of streptococcus mastitis which runs an acute course and is to be distinguished from the widely

prevalent chronic form. Twenty-four cases of the acute form, as it occurred over a period of two years in a herd of 110 cows, is described. Twelve were of such severity that the affected quarter was partially or completely destroyed. Such animals yielded a permanent reduction in milk (7 to 11 pounds daily). The disease appeared to arise spontaneously after an absence of two years. An account also is given of this type of mastitis in three other herds.

STUDIES ON FELINE DISTEMPER. E. Hindle and G. M. Findlay. Jour. Comp. Path. & Therap., xlv (1932), p. 11.

The clinical symptoms of feline distemper are very variable and offer no certain method of clinical diagnosis. Pathologically, there is constantly present some degree of congestion of the small intestines. Enlargement and congestion of the abdominal lymph-glands is generally observed and, in addition, enlargement of the spleen, pleurisy and peritonitis also may occur. A variety of bacteria have been isolated, only eight per cent having been bacteriologically sterile. These bacteria apparently are not etiological factors. Feline distemper can be transmitted by fomites. The disease can be transmitted by Berkefeld filtrates of nasal washings of cats with distemper or by filtrates of the spleen and abdominal glands. The disease can be communicated to cats and carried on in series by intranasal instillations of such filtrates and by subcutaneous, intraperitoneal and intracerebral inoculations. The primary cause is a filtrable virus. Dogs, ferrets, mongooses, rabbits, guinea pigs and mice are insusceptible. The virus may be preserved in 50 per cent glycerin. It is attenuated or killed by drying at room temperature. The inoculation of such dried virus is followed by some degree of immunization. The ingestion of large doses of immune serum protects kittens at least temporarily against inoculations of virus.

CHICKENS DEFINITIVE HOSTS TO SPECIES OF PROSTHOGONIMUS. Olga Lakela. Poultry Sci., xi (1932), 3, p. 181.

Nymphs of dragonflies of four different genera and found to harbor encysted trematodes were fed to five incubator chicks and five incubator ducks. They were killed at intervals to note the development of the flukes. The parasites developed in the bursa Fabricii (located near the cloaca) of both male and female ducks and chicks. The amount of infection in ducks was greater than in chicks because the ducks ate the nymph fragments more

readily, getting a greater number of the cysts. The development of the worms in ducks, however, was retarded. Out of 196 worms recovered, only three were sexually mature; 34 recovered from the chicks after the fifth day were all sexually mature. Segregated cysts were fed to male and female adult birds. In all females, flukes varying in number were collected from the oviducts 17 days later. The examination of the males was negative. The bursa Fabricii was free from infection in all cases. Minute clots of blood were noted in all cases of infection; one bird showed, in addition, a partly formed egg and masses of albumen.

A Virus Disease of Parrots and Parrakeets Differing from Psittacosis. Thomas M. Rivers and Francis F. Schwentker. Jour. Exp. Med., lv (1932), 6, p. 911.

The virus of parrots and parrakeets discovered by Pacheco, Bier and Meyer is unrelated to the agent causing psittacosis either in birds or in man. The virus is fairly species-specific and manifests itself chiefly by the production of areas of focal necrosis in the liver and acidophilic intranuclear inclusions in affected cells. One- or two-day-old chicks are partially susceptible while chick embryos are suitable hosts for the serial passage of the virus. Man is not highly susceptible, as laboratory workers did not contract the disease from birds infected experimentally. The virus can be stored in 50 per cent glycerin and remain viable for at least four months.

Further Researches into the Equine Anemias. A. Krupski. Abst. Vet. Bul., ii (1932), 5, p. 258.

In Switzerland, equine anemia caused by a filtrable virus is uncommon, though anemias of different etiology do occur. The author believes that anemia is often a sequel to infection with strangles and he produces experimental evidence in support of this view. The anemia is the result of impaired hemopoiesis and not the destruction of formed blood cells. The immunization of horses against strangles is discussed.

On the Effect of Intravenous Injection of Glycine on the Serum Calcium. Noah Morris, J. Basil Rennie and Samuel Morris. Brit. Jour. Exp. Path., xiii (1932), 2, p. 132.

The intravenous injection of glycine solution in rabbits and goats leads to a fall in serum calcium. Glycine seems to carry

calcium with it into the tissue cells. When glycine is injected intraperitoneally so as to permit only slow absorption, the serum calcium is not affected. The excess production of amino-acids probably leads to a withdrawal of calcium from the serum into the tissues.

MORTALITY IN NINETY-SIX GROUPS OF CHICKS IN WHICH PULLORUM DISEASE OCCURRED. C. M. Hamilton. Poultry Sci., xi (1932), 3, p. 185.

Ninety-six questionnaires (representing 108,114 chicks), regarding mortality for the first three weeks brooding, were returned to the author. With the exception of two groups, these chicks were hatched from eggs laid by hens which had not been blood-tested for pullorum disease. The chicks were from 55 different hatcheries, were fed 11 different brands of feed and were brooded under 18 different types of brooders. The author brings out the importance of this disease among baby chicks when he concludes that the 26 per cent mortality experienced during the first three weeks brooding in the 96 groups of chicks in which chicks were found to be infected with *Salmonella pullorum* was heavier than the mortality in a limited number of chicks in which brooding trouble was encountered but which were apparently free from pullorum disease.

THE DIGESTION AND ABSORPTION OF RAW STARCH IN DOGS. B. B. Roseboom and J. W. Patton. Amer. Jour. Physiol., c (1932), p. 178.

Two female dogs weighing 15.5 and 17 kilograms, respectively, were given daily injections of phlorizin in oil (1.2 grams). The urine was collected in 24-hour periods. The dogs were fed 250 grams of raw, ground, lean beef daily. When the D/N ratio approached 3.65, after a few preliminary days, the raw corn-starch was added to the ration for one day and determinations continued until the D/N ratio again approached 3.65. The "extra" glucose derived from the starch was calculated. The results show a nearly quantitative digestion of raw starch.

INFECTION OF THE CLOACA WITH THE VIRUS OF INFECTIOUS BRONCHITIS. C. B. Hudson and F. R. Beaudette. Sci., lxxvi (1932), 1958, p. 34.

Cotton swabs infected with the virus of infectious bronchitis have been introduced into the cloacae of birds with the result

that after three days an acute inflammation developed in the proctodeumal portion of this structure. The virus was carried through four generations (from cloaca to cloaca) by means of infected cotton swabs. Birds inoculated intratracheally by swabs from the cloacae of these birds showed typical symptoms of the disease. Tests of immunity showed that birds previously attacked in the cloaca resisted tracheal infection, and those recovered from tracheal inoculation resisted cloacal infection.

SUPERINFECTION OF CATS WITH TAENIA TAENIAEFORMIS. Harry M. Miller, Jr. Jour. Prev. Med., vi (1932), 1, p. 17.

Kittens and cats infected with mature *Taenia taeniaeformis* were not protected against superinfection with *Cysticercus fasciolaris*, the larval stage of this worm, mature and immature worms were present in seventeen individuals, and nineteen control animals had immature worms only. The cysticerci fed failed to become established in the intestines of two cats having mature worms, and in three control animals. Lack of protection has thus been demonstrated for the adult form, which is an intestinal parasite, although the authors have previously shown that the albino rat is immune to superinfection with the larval stage, which is a tissue parasite.

MECHANISM OF EXPERIMENTAL TUBERCULOSIS INFECTION. A Boquet, J. Valtis and A. Saenz. Abst., Arch. Path., xiii (1932), 6, p. 1002.

A careful study of the pathogenesis with regard to the guinea pig indicates that the tubercle bacilli enter by way of the lymphatics, then get into the blood-stream and thus spread to the viscera. The rate of diffusion varies with the method of inoculation and with the number and virulence of the organisms. In massive infection, the bacteriemia appears after an hour and persists until death. Progress is much slower with lighter inocula. Certain tissues, such as the pulmonary and serous parenchyma, owing to the extent of the surfaces and to the multiplicity of potential foci, are more affected than subcutaneous or submucosal connective tissue, accounting for the gravity of pulmonary, pleural or peritoneal infection. From the fact the grass bacillus invades in much the same manner as the tubercle bacillus, except in degree, it is concluded that the fatty structure is more involved than the actual pathogenicity. Actual

virulence depends more on the ability to multiply in the tissues. Thus, with a slight infection with virulent organisms, the bacteriemia may be so delayed that immunity offsets the effects. Localized foci develop, and latent infections may occur, as seen in naturally infected men and animals.

TUBERCLE BACILLI IN THE BLOOD-STREAM OF RABBITS DURING THE COURSE OF INFECTION. Lucy Mishulow and Wm. H. Park. Jour. Prev. Med., vi (1932), 2, p. 95.

From two experiments it seems probable that there is a rapid localization of the tubercle bacilli inoculated into the blood-stream, as shown by the tremendous decrease in their number between 12 and 24 hours after inoculation, and the steady decrease up to the fourth day. The organisms persisted in the blood-stream throughout the entire course of the infection, although they fluctuated in number from day to day. There was a marked rise in numbers on the day of death. The authors conclude that it is justifiable to believe that there is a steady dissemination of the tubercle bacilli from the local lesion into the blood-stream.

SUSCEPTIBILITY OF BATS TO INFECTION WITH THE HORSE TRYPANOSOME, TRYPANOSOMA HIPPICUM (DARLING) IN PANAMA. Lawrence H. Dunn. Jour. Prev. Med., vi. (1932), 3, p. 155.

A number of experiments have demonstrated that bats are readily susceptible to infection with the horse trypanosome, *Trypanosoma hippicum* (Darling), the causative agent of the "murrina" of horses in Panama. The infection was produced in the bats by infected blood being injected intraperitoneally; by passing it through a rubber tube into the stomach; and by feeding it by mouth. When the bats were fed infected blood by mouth, the incubation period was from five to nine days. The course of the disease ranged from 2 to 26 days and usually terminated fatally.

ORGANISMS OF THE B. INFLUENZAE GROUP IN FOWLS. C. A. McGaughey. Jour. Comp. Path. & Therap., xlv (1932), 1, p. 58.

The author has isolated an organism of the influenza group from the upper respiratory tract of fowls. Seven strains were isolated from six birds suffering from inflammation of the respiratory tract which had occurred as an outbreak in a flock.

Four strains were obtained from the larynx of three birds with no abnormality of the upper respiratory tract. One normal fowl did not yield a culture. The morphology and cultural characters are discussed. Some of the strains isolated were pathogenic for mice and possessed invasive properties. The author is in doubt as to the significance of these organisms which, he thinks, can be found in the upper respiratory tract of many normal fowls.

STUDIES IN THE HISTOPATHOLOGY OF LOUPING-ILL. A. Brownlee and D. R. Wilson. Jour. Comp. Path. & Therap., xlv (1932), 1, p. 67.

Lesions of meningo-encephalo-myelitis have been found in all natural cases of true louping-ill. Positive diagnosis of louping-ill was determined by the presence of active louping-ill virus in the central nervous system by inoculation of susceptible animals. No lesions of meningo-encephalo-myelitis were found in cases clinically resembling louping-ill but from which no virus was obtained. Severe damage to the nerve cells of the medulla and all parts of the spinal cord often occurred. Specific inclusion bodies were not demonstrated in the nervous tissues of infected sheep. Intracellular bodies of a non-specific nature were found in the large nerve cells of the spinal ganglia. In the pig the nervous tissues show very intense cellular infiltration and relatively little destruction of nerve cells. In the mouse the principal lesion found was necrosis of the majority of the large nerve cells of the medulla and cord.

BACTERIAL ENDOTOXIN. SEARCH FOR A SPECIFIC INTRACELLULAR TOXIN IN S. PULLORUM. John H. Hanks and Leo F. Rettger. Jour. Immunol., xxii (1932), 4, p. 283.

The cell bodies of *S. pullorum* cultures contain, and by appropriate extraction methods yield, a relatively heat-resistant poison which is highly toxic for rabbits and is capable of killing guinea pigs and mice. This toxin does not cause noticeable symptoms in chicks regardless of the route by which it is introduced. The toxic principle was fairly stable in hydrogen-ion concentration ranging from pH 3.0 to 12.0, and did not deteriorate during exposure to direct sunlight for 24 hours. It was destroyed by the prolonged action of the trypsin or pepsin. It was not dializable through parchment bags, and could be precipitated with ammonium sulfate or acetic acid alcohol. Immu-

nization with toxin filtrate induced tolerance to the toxin, but did not afford protection against subsequent infection with live cultures. Growth or toxin production was not materially increased under tensions of carbon dioxid and oxygen which approximated those of animal tissues. Pullorum disease appears to be a septicemia rather than a toxemia.

PROPERTIES OF THE CAUSATIVE AGENT OF A CHICKEN TUMOR. III. Attempts at isolation of the active principle. James B. Murphy, Ernest Sturm, Albert Claude and Oscar M. Helmer. Jour. Exp. Med., lvi (1932), 1, p. 91.

By two methods a protein fraction can be separated out from a chicken tumor I extract, which carries all of the tumor-producing agent. The precipitate can be dissolved and reprecipitated a number of times without causing loss of activity. The agent can be dissociated largely from the protein, as shown by the fact that aluminum hydroxid will adsorb the protein from an extract and leave the agent behind. This purified material has a very low protein content, if any, shown by both clinical and biological tests.

PROPERTIES OF THE CAUSATIVE AGENT OF A CHICKEN TUMOR. IV. Association of an inhibitor with the active principle. James B. Murphy and Ernest Sturm. Jour. Exp. Med., lvi (1932), 1, p. 107.

The presence of an inhibiting substance in the chicken tumor is shown by the fact that a desiccate of the tumor is more active after it has been washed two or three times with water, and that an extract of the tumor is more potent after some factor is removed by adsorption with aluminum hydroxid. When the tumor-producing factor in an extract of a slow-growing tumor has been destroyed by heating at 55° C., it is found to have the property of neutralizing a highly active tumor extract. This inhibiting property is destroyed by heating over 65° C.

PROPERTIES OF THE CAUSATIVE AGENT OF A CHICKEN TUMOR. V. Antigenic properties of the chicken tumor. James B. Murphy, Ernest Sturm, Giovanni Favilli, Donald C. Hoffman and Albert Claude. Jour. Exp. Med., lvi (1932), 1, p. 117.

The injection of tumor extracts and their active protein fractions into rabbits induced the formation of precipitins and neutralizing antibodies. When the major portion of proteins in the

tumor extract had been eliminated, it induced the formation of neutralizing antibodies, but not of precipitins. The tumor agent, more highly purified by removal of the viscous fraction, did not induce precipitins, and only two out of the fifteen sera gave any evidence of neutralizing bodies. After the removal of the major portion of the protein, the extracts showed insufficient interaction with the sera to fix complement.

METABOLISM STUDIES ON THE BRUCELLA GROUP. IV. The bacteriostatic action of dyes. K. F. Meyer and C. E. ZoBell. Jour. Inf. Dis., li (1932), 1, p. 72.

The advantages of a semisolid medium in tubes with a low protein content and a constant concentration of dye, inoculated with a definite number of cells of Brucella, have been demonstrated experimentally to show the fundamental differences of the various strains of Brucella with an accuracy of 98 per cent. Brucella can be placed into three general groups by recording their tolerance towards thionin, fuchsin and pyronin. The boundaries tend to merge. Atypical strains have been found in a number of instances.

METABOLISM STUDIES ON THE BRUCELLA GROUP. V. The production of hydrogen sulphide. C. E. ZoBell and K. F. Meyer. Jour. Inf. Dis., li (1932), 1, p. 91.

The ability of representatives of Brucella to produce hydrogen sulfid, as a single differential test, is of little value, even when the test is conducted under controlled conditions of inoculation and incubation. In conjunction with the dye-tolerance tests, the ability to produce hydrogen sulfid subclassifies the Brucella group into three major and three minor subgroups.

METABOLISM STUDIES ON THE BRUCELLA GROUP. VI. Nitrate and nitrite reduction. C. E. ZoBell and K. F. Meyer. Jour. Inf. Dis., li (1932), 1, p. 99.

Representatives of Brucella reduce nitrates to nitrites when tested in semisolid media containing 0.2 per cent potassium nitrate. The nitrites also are rapidly reduced. The authors describe differences in the nitrate and nitrite metabolism which, when judiciously employed, may be used in the characterization of the representatives of Brucella. The use of a semisolid nitrate medium for the nitrate reduction test as a standard procedure is advocated.

Regular Army

First Lt. Arvo T. Thompson is relieved from duty at Front Royal quartermaster depot, Front Royal, Va., effective at such time as will enable him to proceed to New York, N. Y., and sail on or about January 12, 1933, for the Philippine Department.

Major Philip H. Riedel is relieved from duty at the San Francisco general depot, Fort Mason, Calif., effective at such time as will enable him to sail on the transport scheduled to leave that port on or about January 28, 1933, for the Panama Canal Department.

Each of the following-named officers of the Veterinary Corps is relieved from his present assignment and duty at the station indicated after his name, effective at such time as will enable him to proceed to New York, N. Y., and sail on or about January 12, 1933, for the Philippine Department:

Major Ralph B. Stewart, Carlisle Bks., Pa.
Captain Frank M. Lee, Fort Benning, Ga.

Major Louis G. Weisman is assigned to Fort Benning, Ga., for duty upon completion of his present tour of foreign service in the Panama Canal Department.

Each of the following-named officers of the Veterinary Corps is assigned to duty at the station indicated after his name, effective upon completion of his present tour of foreign service in the Philippine Department:

Captain Laurence R. Bower to Fort Bliss, Tex.
Captain Elmer W. Young to Front Royal Q. M. D., Front Royal, Va.

Major Jesse D. Derrick is assigned to duty at Fort Reno, Okla., and to additional duty at the purchasing and breeding headquarters, that station, effective upon completion of his present tour of foreign service in the Philippine Department.

So much of par. 24, S. O., 202, W. D., 1932, as directs 2nd Lt. Richard G. Yule to report to the commanding officer Army Medical Center for duty for the purpose of pursuing a course of instruction at the Army Veterinary School, is amended so as to direct him to report to the commanding officer Army Medical Center for duty.

The promotion of Captain John R. Ludwigs to the grade of major, to rank from September 26, 1932, is announced.

Veterinary Reserve Corps

Promotions

		To	
Embree, Warren Jesse	Lt. Col.	602 Livestock Exchange Bldg., Union Stock Yards, Chicago, Ill.
Kitzhofer, Joseph H.	Capt.	332 Post Office Bldg., Oklahoma City, Okla.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Proceedings of the Sixty-ninth Annual Meeting, Atlanta, Georgia, August 23 to 26, 1932

(Continued from page 537, October issue)

Section on General Practice

WEDNESDAY MORNING, AUGUST 24, 1932

The first session of the Section on General Practice was called to order at 9:30 a. m. by Dr. M. R. Blackstock, Chairman. (Dr. J. Lee Hopping, Secretary.)

CHAIRMAN BLACKSTOCK: The Chairman of this Section is supposed to make an address. Just how lengthy this should be I do not know. There has not been much in the records of the past concerning this address, but I want to say this much.

First, in the capacity of Secretary of the Southern States Veterinary Medical Association, I want to add to that which has already been said in assuring you that the welcome you have been given is genuine and comes from the entire veterinary population of the Southeast.

The Atlanta boys have done some real hard work, as they promised to do, when you were invited to come south at the Kansas City meeting one year ago. They have had at least the moral support of the men in these nine southeastern states. Now they all stand ready to do anything in their power to make this a most profitable and enjoyable time for you and your families.

We are all especially thankful and proud that the weather man has been so kind in sending just the sort of weather we have received, and we hope that it may last a few more days. When this meeting comes to an end, we hope that you may honestly say that this has been a most outstanding and enjoyable meeting.

* * * *

The following program was presented:

"Value of Systemic Alkalinization in Influenzal Types of Disease," Dr. R. W. Hixson.

"Lameness and Its Treatment in the Horse," Dr. John Baird.

"Idiopathic Hemorrhagic Hepatitis of Swine" (Illustrated), Drs. A. H. Quin, Jr., and J. D. Shoeman. (Read by Dr. Quin.)

"Sterility in Cattle," Dr. W. A. Barnette.

. . . The session adjourned at 1 p. m. . . .

RECESS

THURSDAY MORNING, AUGUST 25, 1932

The second session was called to order at 9:30 a. m. by Chairman Blackstock.

* * * *

The following program was presented:

"The Correlation Between Certain Properties of the Milk and the Type of Inflammation in Acute Mastitis," Drs. Ralph B. Little and F. S. Jones. (Read by title.)

"Udder Diseases in Cattle," Dr. D. H. Udall.

"Salt Sick: Its Cure and Prevention in Cattle" (Illustrated), Dr. A. L. Shealy. (Read by Dr. W. N. Neal.)

"The Present Status of Anthelmintic Medication for Gastro-intestinal Parasites of the Horse," Dr. Willard H. Wright.

"Pasture Diseases of Sheep," Dr. W. H. Lytle. (Read by title.)

"Ergotism of Cattle in Kansas," Dr. J. W. Lumb. (Read by title.)

. . . The session adjourned at 12:15 p. m. . . .

ADJOURNMENT

Section on Sanitary Science and Food Hygiene

WEDNESDAY MORNING, AUGUST 24, 1932

The first session of the Section on Sanitary Science and Food Hygiene was called to order at 9 a. m. by Dr. W. K. Lewis, appointed to act as Chairman in the absence of Dr. W. H. Lytle. (Dr. J. V. Knapp, Secretary.)

* * * *

The following program was presented:

"Establishing Accredited Abortion-free Herds," Dr. C. A. Cary.

"The Relation of the Veterinary Profession to Municipal Meat Inspection," Dr. E. D. King, Jr.

"Municipal Dairy Inspection," Dr. W. D. Martin.

"Edward Jenner, the Father of Vaccination" (Illustrated), Dr. C. N. McBryde.

. . . The session adjourned at 12 noon. . . .

RECESS

THURSDAY MORNING, AUGUST 25, 1932

The second session was called to order at 9 a. m. by the Secretary, Dr. J. V. Knapp, acting Chairman in the absence of Dr. Lewis.

ACTING CHAIRMAN KNAPP: In the preparation of this program Dr. Lytle and I attempted to present something that would be of national interest, and yet have the program talent come as much as possible from the Southeast. I found it just as difficult to get people to appear on the program of an American Veterinary Medical Association meeting as I do for our State Association. That may seem strange, because the honor of appearing before the American Veterinary Medical Association ought to appeal to all veterinarians, but we had quite some difficulty in getting people here.

We thought that we would present a program which the whole Association might take back. For instance, it is our opinion that meat and milk inspection possibly offers the veterinarian one of the largest fields, and we wanted to give some information along that line. Some of the new phases of the work coming to our attention, particularly in the field of sanitary science, such as anaplasmosis and anthrax vaccination, were thought of. Unfortunately we are not able to get anyone here who is carrying on anaplasmosis experiments. The Bureau of Animal Industry could not let their men come on account of the expense. The work in Florida had not gone far enough to be of any real value.

* * * *

The following program was presented:

"Handling of Anthrax," Dr. C. A. Cary.

"Blackleg Aggressin Immunization" (Illustrated), Drs. John Reichel and J. E. Schneider. (Read by Dr. Reichel.)

"Veterinary Medicine in Mexico," Dr. Francisco Moguel M.

. . . The session adjourned at 12 noon. . . .

ADJOURNMENT

Section on Research

WEDNESDAY MORNING, AUGUST 24, 1932

The first session of the Section on Research was called to order at 9 a. m. by Dr. W. A. Hagan, Chairman. (Dr. C. C. Palmer, Secretary.)

CHAIRMAN HAGAN: The first thing on the program is the address of the Chairman. The idea of the Chairman is that no address is called for. I have nothing to report. The Secretary has done all the work in getting up the program. We will now have the report of the Secretary.

SECRETARY PALMER: The Secretary's report is very much like that of the Chairman. My report is printed in the program, so I do not think it is necessary for me to make any further report.

CHAIRMAN HAGAN: We will assume that the reports of the Chairman and Secretary have been accepted, and we will proceed with the remainder of the program.

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The following papers were presented:

"A Study of Bovine Coccidiosis: II," Dr. I. D. Wilson.

"Studies on Canine Distemper: III. A Comparison of Natural and Experimental Virus Infections," Dr. A. S. Schlingman.

"Some Observations on the Pathology of Johne's Disease," Drs. E. T. Hallman and J. F. Witter. (Read by Dr. Witter.)

"Further Studies of Natural Brucella Infection in Swine," Drs. Howard W. Johnson, I. Forest Huddleson and E. E. Hamann. (Read by title.)

"A Study of Brucella Infection in Swine and Employees of Packing Houses," Drs. I. Forest Huddleson, Howard W. Johnson and E. E. Hamann. (Read by title.)

"A Continuation of the Study of the Etiology of Infectious Diarrhea (Winter Scours) in Cattle," Drs. F. S. Jones and Ralph B. Little. (Read by title.)

"Relation of the Alcohol-Milk Test to Pregnancy in Cattle," Dr. C. C. Palmer. (Read by title.)

. . . The session adjourned at 12 noon. . . .

RECESS

Section on Poultry

WEDNESDAY MORNING, AUGUST 24, 1932

The first session of the Section on Poultry was called to order at 9 a. m. by Dr. Frank Thorp, Jr., Chairman. (Dr. M. W. Emmel, Secretary.)

CHAIRMAN THORP: I have no formal address to make. I will not bother you with that. There is just one point I want to mention. I may not be able to recall all of your names. Dr. Hoskins requests that each speaker, in the discussion, give his name, so that we may have it for our records. Next we will hear the Secretary's report.

SECRETARY EMMEL: Gentlemen, I told Dr. Thorp that my speech would be as short as, if not shorter than, his; so I refrain from making a report.

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The following program was presented:

"A Preliminary Report on the Study of Poultry Vermifuges," Dr. E. F. Thomas.

"So-Called Range Paralysis of the Chicken," Dr. F. D. Patterson, H. L. Wilcke, Dr. Chas. Murray and E. W. Henderson. (Read by Dr. Patterson.)

"Blood Studies of Fowls with Various Forms of Lymphomatosis (Fowl Paralysis)," Dr. E. P. Johnson and Betty V. Conner. (Read by Dr. Johnson.)

"Observations on the Use of Pigeon-Pox Virus as a Cutaneous Vaccine for Fowl-Pox in an Egg-Laying Contest," F. H. Orr, Jr., and Dr. M. W. Emmel. (Read by Dr. Emmel.)

"On the Disinfection of Avian Fecal Material," Drs. W. L. Mallman and W. L. Chandler. (Read by Dr. Emmel.)

. . . The session adjourned at 12 noon. . . .

RECESS

Section on Research and Section on Poultry Joint Session

THURSDAY MORNING, AUGUST 25, 1932

The second session of the Section on Research and the second session of the Section on Poultry were convened as a joint session of the two sections, at 9 a. m., Dr. Frank Thorp, Jr., Chairman, presiding.

* * * *

The following program was presented:

"A Discussion of Some Fundamental Principles and Practices Underlying the Application of the Agglutination Tests for Bang's Disease," Drs. C. P. Fitch and C. R. Donham. (Read by Dr. Fitch.)

"A Liver Function Test in Sheep," Dr. J. N. Shaw. (Read by Dr. B. T. Simms.)

"Treatment for Mastitis with Ultra-Violet Light, Formalin, Colloidal Carbon and Autogenous Bacterin," Dr. E. L. Gildow, H. C. Hansen and V. A. Cherrington. (Summary read by Dr. Simms.)

"Infections of Fetuses and Foals," Drs. W. W. Dimock and P. R. Edwards. (Read by title.)

"Experiments on Fowl-Pox," Dr. C. L. Martin. (Read by Dr. M. W. Emmel.)

"Further Studies on the Relative Efficiency of Vermifuges for

Poultry," Dr. Wm. L. Bleecker and Robert M. Smith. (Conclusion read by Dr. Emmel.)

"The Pathogenicity of the Saprophytic Acid-Test Bacilli," Drs. William A. Hagan and Philip Levine. (Read by Dr. Hagan.)

"Some of the Practical Problems of a Poultry Practitioner," Dr. H. J. Seaman. (Read by title.)

. . . The session adjourned at 12 noon. . . .

ADJOURNMENT

Section on Small Animals

WEDNESDAY MORNING, AUGUST 24, 1932

The first session of the section on Small Animals was called to order at 9:30 a. m. by Dr. C. F. Schlotthauer, Secretary, presiding during the temporary absence of Dr. F. W. Morgan, Chairman.

ACTING CHAIRMAN SCHLOTTHAUER: Dr. Morgan has a dental appointment this morning and has not come yet. We will let the Chairman's report go over until tomorrow. I have prepared a short report, but I do not know that it is worth while to present it to this group for it does not really pertain to the group present, but I will present it.

. . . Dr. Schlotthauer read his report. . . .

SECRETARY SCHLOTTHAUER: The program for the Section on Small Animals is one of the most important of this Association. It is your program. It belongs to all of you, yet, year after year, you persist in putting the responsibility of the program on a few. Every man here is capable of preparing and presenting a very worthwhile paper. You all know what you are most interested in, and therefore should submit a paper on that subject, or recommend to the Secretary someone whom you think can do better.

This year we have eight papers listed on our program and two that are not listed. That is all the papers your Secretary could obtain. Gentlemen, that is a sad commentary on our profession. I have visited recently with secretaries of other societies, including some of the medical societies. They informed me that they solicit only special papers. Their programs, otherwise, are made up entirely of papers submitted to the Secretary without solicitation. A committee then selects those papers that are thought to be of greatest value. In this way the programs frequently can be concentrated on a single subject. It is my earnest opinion that we can do the same if we try.

As soon as your new Chairman and Secretary are appointed,

write and inform them what you would like to see presented and also whom you would like to have on the program. If you do this, it will give your Chairman and Secretary a better opportunity to concentrate on certain subjects. And, of course, most important of all, it will be your program. The subjects presented will be those of greatest interest to all. And that, of course, is the desired function of this section.

ACTING CHAIRMAN SCHLOTTHAUER: Is there any comment or suggestion? Everybody should talk—talk here and not when you get home from the meeting. Do your criticizing here and let everyone hear it.

DR. J. L. RUBLE: That is the same thing that exists with the M. D.'s. In arranging the Small Animal Clinic, I understand a great many of the men wanted to be paid to come down here and appear on the program. That is certainly a different chain of thought from that which I have, but I understand men offered to come, provided we paid railroad fare, hotel bill, and everything else.

ACTING CHAIRMAN SCHLOTTHAUER: That is a peculiar thing. I think most state societies do pay expenses but the American Medical Association and all national societies do not. The contributors pay their own expenses. Associations do not pay expenses except in isolated cases in which they want some particular man and he has to come at great expense. Then his expenses are defrayed. Otherwise, all national societies do not pay expenses.

DR. H. J. MILKS: We do not pay expenses in New York State, if the speakers are local men. If we want someone of national reputation and bring him from the outside, we pay him, but otherwise it is as much his job as it is anyone's. I do not see any reason for paying anybody's expenses. If we have a man from some other place—a noted man—it is different. I have had experience in getting up programs for different things, and I know it is hard to get anybody to come in. Men are modest. The men who do prepare papers frequently dislike to volunteer.

ACTING CHAIRMAN SCHLOTTHAUER: If anyone has a suggestion now is the time to make it, because you can all carry it home to your community and talk to your small-animal men and convey these suggestions from the Section in regard to the preparation of future programs. If there is nothing further, we will have the first paper.

* * * *

The following program was presented:

"Critical Tests of the Efficacy of Single Treatments with Tracheal Brushes in the Mechanical Removal of Lung Worms from Foxes," Drs. K. B. Hanson and Frank G. Ashbrook. (Read by Dr. J. E. Shillinger.)

"Intravenous Therapy," Dr. E. J. Frick. (Dr. F. W. Morgan took the chair.)

"Black Tongue," Dr. H. Calvin Rea.

"Ethical Commercialism," Dr. I. M. Hays.

. . . The session adjourned at 12:10 p. m. . . .

RECESS

THURSDAY MORNING, AUGUST 25, 1932

The second session was called to order at 9:30 a. m. by Chairman Morgan.

CHAIRMAN MORGAN: It gives me a great deal of pleasure to see so many of you interested in the Section on Small Animals, and particularly to see the faces of representatives of so many different states. I am not going to burden you with a lengthy address, as we have too many valuable papers and our time is too limited. I am sure you would much prefer to get down to real business rather than to listen to a welcoming address, a lot of windjamming and stuff that is of no material benefit.

Therefore, I will say again, I am proud to have you here with us in the South, and I am proud to be honored with the office of Chairman of the Section on Small Animals. I am sure you will go away from here with many new thoughts and ideas which I hope will prove helpful. We will now proceed with the program.

* * * *

The following papers were then presented:

"Bulla osteotomy in the Cat," Drs. J. E. McCoy and G. W. McNutt. (Read by Dr. Schlotthauer.)

"The Hospitalization and Care of Cats," Dr. H. W. Brown. (Read by Dr. Schlotthauer.)

"Some Cases of Diabetes," Dr. H. J. Milks.

"The Preoperative and Postoperative Care of Small Animals," Dr. C. F. Schlotthauer.

. . . The session adjourned at 12 noon. . . .

ADJOURNMENT

Section on Military Medicine

THURSDAY MORNING, AUGUST 25, 1932

The session of the Section on Military Medicine was called to order at 9 a. m. by Major R. A. Kelser, Acting Chairman in the absence of Major Burlin C. Bridges, Chairman. (Lt. Col. D. M. Campbell, Secretary.)

ACTING CHAIRMAN KELSER: Gentlemen, I have been asked to preside as Chairman, in the absence of Major Bridges. In so far as any address by the Chairman is concerned, we will dispense with that, and will have the report of the Secretary, Lt. Col. D. M. Campbell.

. . . Lt. Col. Campbell read his report. . . .

Report of Secretary

MR. CHAIRMAN AND MEMBERS:

The writer finds himself in doubt as to what to present to the Section in an annual report at this time. Obviously there is no precedent to follow. Since this is the initial meeting of the Section, no report can be made of action taken in response to directions or resolutions of the Section, or upon any matters referred to this office by the Section. Since your Secretary has performed no duties in connection with this Section other than arranging the program about to be presented to you, the question arises: Why make any report at this time?

The reasons for a report seem obvious to me, but under the circumstances, perhaps require some mention. If this section of the A. V. M. A. is to justify itself and achieve a permanent status, it must do so on its record. The Executive Board of the A. V. M. A. created this Section, at its meeting in Chicago, last December, on trial only, the understanding being that it would obtain for two years. If it justified itself in that time by its achievements, it will be continued; if not, it will be discontinued. Hence, the importance of a complete record of its proceedings. The future existence of the Section depends upon it. The present report, it is believed, is necessary to completory.

Furthermore, any action that this Section may see fit to take in the future should be supported by a complete record of its antecedents, that its motives and its importance may be judged intelligently by those whom it is desired to influence. In other words, the history of the Section from its beginning, which was unofficial, should be devoid of lapses. Recording that history cannot begin earlier than the present moment. In my opinion it should not be postponed to a later date.

As to just what groups or individuals this Section owes its remote beginning, the record is somewhat obscure. In the years immediately following the close of the World War, there were several rather informal gatherings of former veterinary officers, at which the advantages of a national organization were discussed, but as far as the records show, on only two occasions were measures taken looking to that end. The first occurred at Chicago, December 3, 1919, when the Society of World War Veterinarians was founded by a group of former veterinary officers meeting for that purpose at the Hotel LaSalle. Membership was limited to veterinarians who had served in any capacity in the Army during the World War. L. A. Merillat, Orrville,

Ohio, was elected National Commander; J. V. Lacroix, Evanston, Illinois, Assistant Commander; and A. A. Leibold, Chicago, Illinois, adjutant. An account of this meeting may be found in *Veterinary Medicine* for January, 1920. No subsequent meeting appears to have been held by this organization.

The second attempt to form a national veterinary association with military aspects occurred at the same place, four years later. In December, 1923, thirty-two officers of the Veterinary Corps of the Regular Army and Veterinary Reserve officers met for a dinner at the Hotel LaSalle in Chicago. The meeting was in response to an invitation of Lieut. Col. W. P. Hill, Corps Area Veterinarian of the Sixth Corps Area. The meeting was a highly successful one. An excellent program of speeches was enjoyed by everyone present. Col. Hill presided; addresses were made by Col. Keck, in charge of Reserve Officer affairs for the Sixth Corps Area; Major Christie, Chief of Staff of the 86th Division, a reserve organization; Capt. MacConachie, late of the Canadian Veterinary Service; Dean David S. White; N. S. Mayo; L. A. Merillat and others. The meeting ended with the organization of the Society of Military Veterinarians, to which veterinary officers, both regular and reserve, and former veterinary officers were eligible for membership. Col. Hill was elected temporary chairman, and Doctor Merillat temporary secretary. The new organization did not prosper. Many of those eligible to membership were indifferent, and a few were actively hostile to it. With the removal of the Army Veterinary School to Washington, which occurred soon afterwards, Col. Hill, who was at its head, was transferred there also and his active support of the new organization in the Middle West was lost. The Veterinary Reserve at that time was in its early developmental stages. There was much misunderstanding of its purpose among former veterinary officers. The idea of an organization to promote its interests slumbered but did not die.

Veterinary Medicine for August, 1930, carried an announcement that there would be a meeting of veterinary officers, both regular and reserve, in connection with the 67th annual meeting of the A. V. M. A., at Los Angeles, in August of that year. Being in Europe at the time, I did not attend the meeting, but am informed a meeting was called at the instance of Major J. G. Townsend, Vet-Res., and was attended by about a dozen, chiefly veterinary officers of the Regular Army. The discussions were informal and related chiefly to the preparation of a program for an officers' meeting in connection with the Kansas City meeting of the A. V. M. A. last year. Major Townsend, Los Angeles, acted as chairman of the meeting. The only published reference to it that I have found appears in the JOURNAL of the American Veterinary Medical Association, December, 1930, and simply states that a meeting was held, to prepare a program for a meeting of veterinary officers to be held in connection with the 1931 convention of the A. V. M. A. at Kansas City, and that N. S. Mayo, J. A. McCallum and Wm. G. Keehn were selected to carry out the wishes of the meeting.

The minutes of the meeting of veterinary officers in Kansas City you have already heard. Its success was undoubted. All three of the papers referred to in the minutes have been published in veterinary magazines of general circulation. Since the meeting was not an official part of the American Veterinary Medical Association convention, in connection with which it was held, and therefore the official journal of the Association was under no obligation to publish them, it may fairly be inferred that the papers constituting the program were published because of their merit. I regard the paper by General Moore as being the most noteworthy contribution to veterinary science

of the year. Major Koon's paper may be found in the July, 1932, issue of the *Veterinary Bulletin*, Doctor Campbell's in the January, 1932, issue of the *North American Veterinarian* and General Moore's in the March, 1932, issue of *Veterinary Medicine*. Copies of these papers, together with a copy of the proceedings of the Kansas City meeting, have been assembled in a loose-leaf binder and placed in the archives of this Section. The papers and proceedings of this meeting, together with the names of those in attendance, will be added likewise.

It is believed that the records of this Section might contain, with advantage, a list of the appointments, promotions, notable achievements, transfers, retirements and deaths of officers of the Veterinary Corps of the Regular Army and National Guard, and of appointments, promotions, assignments to active duty for training and to special service schools of officers of the Veterinary Reserve. Such a list for the last fiscal year has been included in this report.

Because no appointments to the Veterinary Corps of the Regular Army have been made during the year, to fill vacancies that have occurred, the Corps is now short seven of its authorized quota of 126 officers.

The Veterinary Reserve showed some gain during the fiscal year ending June 30, 1932, the total number of officers at that time being 1,020. The preceding year the number was 978. The gain, however, is more apparent than real, since the number on the "ineligible list" also increased; the number on June 30 having been 292. Officers on the ineligible list are in the process of loss to the reserve. They cannot be assigned to active duty training, promoted or recommissioned for more than one five-year period. If they be deducted from the number in the Reserve, as they must be in the course of a few years, because of the expiration of their commissions, and if the 151 officers holding dual commissions in the National Guard and the Veterinary Reserve also be deducted, since they would not be available to the Reserve in case of another war, we have but 577 officers left. The General Mobilization Plan of the War Department calls for 1,647 Veterinary Reserve officers This is far from a gratifying showing for the veterinary profession. The number of veterinary reserve officers (less National Guard officers) now on the eligible list in the various grades are: colonels, 1; lieutenant colonels, 23; majors, 72; captains, 108; first lieutenants, 67; second lieutenants, 297. The accessions to the Corps from the R. O. T. C. during the year were 56 second lieutenants. To quote from "The Veterinary Service of the A. E. F.":

> The duty of the veterinary profession of the United States today is to get sanely behind national defense so that in any future call to duty the animal transport service of the army will have a veterinary corps and a remount service ready with well-conceived rules and regulations to supply motility to troops in action, without which heroism and bayonets are useless pretensions.

A notable contribution to military veterinary medicine during the present year is the publication of "The Veterinary Service of the A. E. F.," by Dr. L. A. Merillat. This historical account is appearing serially in the *North American Veterinarian* and will be published in book form soon. Since the official history of the Medical Department in the World War contains less than three pages concerning the veterinary service of the army, and no history of the veterinary service of the American Army during any other war has ever been published, Doctor Merillat's history constitutes the only source, after the present officers with World War experience have retired, from which veterinary officers and officers of the arms and other services may learn from the experiences of the veterinary service in the World

War. The failure to include a volume on the veterinary service in "The Medical History of the World War" was a serious omission. There is no officer in the Veterinary Reserve, seriously endeavoring to qualify himself for the duties of his position, and probably none in the veterinary service of the Regular Army, but finds himself handicapped by the lack of such information. The need of officers of the line and of the Quartermaster Department for such information differs only in degree from that of veterinary officers. Doctor Merillat's history will in part fill this need. If it shall induce some one to supply a history of the same service in the United States for the same period, it will have doubly served the profession, the army and the nation.

(Signed) D. M. CAMPBELL

ACTING CHAIRMAN KELSER: You have heard the report of the Secretary. Unless there is some discussion or objection, it will be accepted and adopted.

* * * *

The following program was presented:

"The Practicing Veterinarian and His Relationship to the Army," Lt. Col. D. S. Tamblyn. (Read by Lt. Col. A. E. Cameron.)

"How Can the Operation of the Army Veterinary Service Be Improved in Future Engagements in the Light of Experiences of the World War?" Major R. A. Kelser.

"Reminiscences of the World War," Dr. L. A. Merillat. (Read by Lt. Col. D. M. Campbell.)

"Some Lessons of Peace," Col. N. S. Mayo.

"Training Policies for Veterinary Reserve Officers," Dr. J. G. Horning. (Read by title.)

. . . The session adjourned at 12 noon. . . .

ADJOURNMENT

Special Pig Diseases Number

The October, 1932, issue of the *Veterinary Journal* (London) was devoted entirely to articles dealing with various diseases of swine, including asthenia, tuberculosis, swine fever (hog cholera), swine erysipelas, anemia in pigs, helminthiases, and other conditions. Several articles have been contributed by veterinarians of Continental Europe on disease conditions among swine in their respective countries. Other related subjects discussed include the feeding of pigs, bacon factory inspection, and the electric stunning of pigs for slaughter. Sir Frederick Hobday contributed an article entitled "A Preliminary Note on the Use of the Electro-Lethaler for Anesthetizing Pigs and Other Wild and Troublesome Patients."

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The forty-eighth semi-annual meeting of the Veterinary Medical Association of New Jersey was held at the Chelsea Hotel, Atlantic City, July 14-15, 1932.

Papers presented during the sessions included the following:

"The Importance of Milk Inspection in Relation to Public Health," by Dennis J. Sullivan, Jersey City.

"Clinical Diagnosis in Bovines," by Dr. E. R. Cushing, Plainfield.

"Diseases of the Teeth and Eyes of Canines," by Dr. E. R. Blamey, New York City.

"Some Observations in Small Animal Practice," by Dr. Raymon M. Staley, Narberth, Pa.

"Ano-Vulvitis in Cattle," by Dr. R. B. Little, Princeton.

"The Contrastive Diseases of Man and the Lower Animals," by Dr. Herbert Fox, Philadelphia, Pa.

"Surgical Operation for Roaring," by Dr. William J. Lee, Philadelphia, Pa.

"Intravenous Therapy," by Dr. E. B. Dibbell, Baltimore, Md.

The clinical features provided special interest for those in attendance. In addition to their papers, Dr. Blamey conducted a clinic on oral and ophthalmic surgery of small animals; Dr. Lee demonstrated his method for surgical relief of roaring, and Dr. Dibbell showed his methods of intravenous anesthesia and medication and other operations on small animals, including bandaging, reduction of fractures and the use of the traction splint.

Members of the Association also contributed to the clinics. Dr. R. E. Mosedale performed an ovariectomy, and Dr. C. J. McAnulty discussed and demonstrated the proper technic for tail-cutting and the trimming of toe-nails of dogs.

Four applicants were admitted to membership, making a total of fourteen new members for the year.

The attendance of 78 was the best for several years at a summer meeting. The Local Committee, consisting of Drs. C. J. McAnulty, Chairman, and Louis Goldberg, Frederick Stehle and

J. A. Webb, all of Atlantic City, and A. D. Goldhaft, of Vineland, did a splendid piece of work in arranging the program and providing facilities for the clinics.

J. G. HARDENBERGH, *Secretary.*

VETERINARY ASSOCIATION OF SASKATCHEWAN

The annual meeting and summer school of the Veterinary Association of Saskatchewan was held at the University of Saskatchewan, July 26, 1932.

For the last twenty years it has been the custom of the Association to bring in some outstanding authority to conduct the scientific part of the meeting and the clinic. This year, the following members of the Association undertook to provide the scientific papers and conduct the clinic: Drs. W. Eason, Watrous; L. J. Hewitt, Regina; M. Barker, Regina; J. L. Millar, Asquith, and Prof. J. S. Fulton, Saskatoon.

The attendance was larger than had been expected. In addition there were six visitors from various parts of Canada and the United States. The morning session was devoted to business, reports of committees, reading of the annual audit, election of members of the Council and a keen discussion of the affairs of the Association in particular and the veterinary profession in general.

The President, Dr. A. Chambers, opening the discussion, surveyed the status of the veterinary profession in Saskatchewan during the past year. He pointed out that it was gradually losing ground, due partly to the mechanization of the farms, repeated crop failures in the most fertile part of the Province, also partly to the inertia and lack of leadership in various communities in matters concerning the health of animals and public health, which a veterinary surgeon should give. Due to this latter factor, the professional work that the veterinary surgeon should do was being taken over by the technical agriculturists, medical men and the laity. In so far as the veterinary surgeon was at fault, the remedy lay with himself. He should be active in all matters concerning the health of live stock, including poultry. The relationship between public health and animal diseases should be brought before the public as occasion demanded. Meat and milk inspection, tuberculosis, undulant fever, septic sore throat from milk, and many other relative subjects could all be dealt with by the veterinary surgeon to the great benefit of the community and his own professional stand-

ing. These matters could be dealt with in addresses over the radio before community clubs and agricultural societies.

After lunch, which was provided in the University dining-hall, the annual visit was made to the Veterinary Memorial in Convocation Hall. The handsomely-cut bronze plate was erected in memory of members of the Association who gave their lives in the Great War of 1914-1918. The afternoon session was devoted to the reading of scientific papers and a surgical clinic.

The program follows:

"Navel-Ill in Foals."
"Actinomycosis," by Dr. W. Eason, Watrous.
"Bovine Practice," by Dr. L. J. Hewitt, Regina.
"Veterinary Science in Saskatchewan, Present Status and Future Needs," by Dr. J. S. Fulton, Saskatoon.
"Meat and Milk Inspection for Rural Communities," by Dr. M. Barker, Regina.
Clinic in Animal Pathology Laboratory.
"Spaying Heifers," by Dr. J. L. Millar, Asquith.
"Spaying in the Cat," by Dr. D. W. McDonald, Moose Jaw.

At the close of the meeting a vote of thanks was passed to the President of the University for the accommodation provided and for his unfailing interest in the welfare and progress of veterinary science. After the meeting, members expressed the opinion that they had gotten more out of the School this year than in any previous year.

NORMAN WRIGHT, *Secretary.*

WYOMING VETERINARY ASSOCIATION

On September 14, 1932, thirteen Wyoming veterinarians met at the Gladstone Hotel in Casper, Wyo. This group, composed of practitioners, state and B. A. I. men, organized the Wyoming Veterinary Association, the first veterinary association in the State.

The business of organization was accomplished at the morning session of the meeting. The following officers were elected: President, Dr. G. D. Anderson, Torrington; vice-president, Dr. R. Blackner, Lyman; secretary-treasurer, Dr. H. D. Port, Cheyenne.

The afternoon session was given over to a discussion of veterinary problems in Wyoming. Dr. Anderson spoke on "Encephalomyelitis in Horses," which disease has caused large losses in his territory. Dr. J. B. Fuller, of Huntley, outlined his experience with the disease. Dr. G. H. Good, deputy state veterinarian, discussed his recent investigations of a disease prevalent in northern Wyoming which, with the coöperation of

the University of Wyoming, was found to be caused by a grass-borne fungus.

Dr. A. G. Fisk, of Denver, Colo., a past president of the Colorado Veterinary Medical Association, was elected an honorary member of the Wyoming Association, in recognition of his helpful suggestions on its organization. The members of the newly-formed association decided to accept an invitation of several years' standing to affiliate with the Colorado Association.

Not all the veterinarians of the State were able to attend the meeting, but the absentees sent regrets and expressed a desire to join the Association. It is expected that the membership will be increased to about twenty in the near future.

C. E. WILMOT, *Resident Secretary.*

SOUTHEAST KANSAS VETERINARY MEDICAL SOCIETY

Twenty-one veterinarians and their families attended the meeting of the Southeast Kansas Veterinary Medical Society at Independence, September 16, 1932. Dr. H. G. Stephenson, of Independence, entertained the Society at his hospital in the afternoon, where a small-animal clinic was conducted by Dr. Chas. W. Bower, of Topeka.

Following the clinic, Dr. J. H. Weiner, of Kansas City, gave a very interesting paper on blood chemistry, outlining the value of the use of glucose and calcium in veterinary therapy. Dr. S. J. Schilling, of Kansas City, discussed the diagnosis and post-mortem findings of swamp fever. Dr. C. A. Pyle, of Sedan, gave a comprehensive report on his findings with the mercuric chlorid test, in the diagnosis of swamp fever, on blood serum sent to him by veterinarians of southeast Kansas.

Dr. S. R. Johnson, of Kansas City, discussed the importance of biological therapy in veterinary practice. Mr. Thomas Dalton, of Topeka, gave a very clear outline of the importance of milk inspection, emphasizing the fact that the veterinarian is the most capable inspector. Dr. W. R. Barnard, of Belleville, president of the Kansas Veterinary Medical Association, gave a very informative talk on the value to the practitioner of the interstate associations.

After the program a large basket dinner was enjoyed by all present. This was followed by a varied vocal and instrumental program.

L. F. BARTHELME, *Secretary.*

KANSAS CITY VETERINARY ASSOCIATION

The Kansas City Veterinary Association held its regular monthly meeting Tuesday evening, September 20, 1932, at the Baltimore Hotel. Thirty members and visitors attended. The program was devoted to the discussion of diseases of cattle incident to shipping, with special attention to the handling of animals sick soon after arrival on the farm.

Dr. R. Meier, of Smithville, Mo., Dr. F. H. Suits, of Odessa, Mo., and Dr. H. J. Hearrington, of Lexington, Mo., gave talks on their experiences in this kind of work. A good discussion followed.

J. D. RAY, *Secretary.*

CENTRAL MISSOURI VETERINARY MEDICAL ASSOCIATION

The Central Missouri Veterinary Medical Association held its regular fall meeting Friday evening, September 23, 1932, at the farm of Dr. I. E. Blosser, three miles east of Malta Bend, Mo. The meeting was scheduled to begin at 6 p. m., with a picnic dinner on the lawn. This was a most successful part of the program, and the food was especially prepared for the non-vegetarian.

Following the supper the ladies retired to the parlors and spent the evening playing bridge, while the veterinarians in attendance met and discussed their local problems pertaining to general practice. The meeting was conducted in the form of a round-table discussion. Dr. L. H. Beebe, of Warrensburg, is president of the Association, and Dr. Geo. F. Townsend, of Sedalia, is acting secretary. The meeting adjourned, after a most enjoyable evening, until the regular spring meeting, which will be held in Sedalia, the date to be determined later.

J. D. R.

NORTHWESTERN MISSOURI VETERINARY MEDICAL ASSOCIATION

The Northwestern Missouri Veterinary Medical Association held its regular quarterly meeting with Drs. C. M. Cline and K. Sears, at Maryville, Mo., October 11, 1932. Twenty-two veterinarians attended the meeting.

The program began about 10:30 a. m., and the entire day was devoted to a clinic, which was held in the local sales pavilion. Many interesting surgical cases were presented, as well as several cases for diagnosis.

A short business session was held, at which time the following officers were elected for the ensuing year: President, Dr. J. L. Cherry, Tarkio; vice-president, Dr. H. B. Allen, Oregon; secretary-treasurer, Dr. R. L. Cundall, Fairfax.

J. D. R.

Condemnations for Tuberculosis Greatly Reduced

Less than one-third as many cattle slaughtered under federal meat inspection, during the year ended June 30, 1932, were affected with tuberculosis as were found in a similar period ten years ago. The total number of cattle inspected was practically the same, being 7,793,878 in the fiscal year 1932, and 7,795,323 in 1922. Cattle showing evidence of tuberculosis in 1932 numbered 38,446 as compared with 146,945 ten years ago.

Since the findings of the federal meat-inspection service furnish an index to the health of market live stock, the comparative figures show the effectiveness of the nation-wide campaign to eradicate bovine tuberculosis. Officials of the U. S. Bureau of Animal Industry have pointed out the similarity between the results of field surveys and federal meat-inspection records. In both cases the proportion of tuberculous cattle in 1932 was found to be about one-third that of ten years ago.

Another significant observation made by veterinarians in the federal meat-inspection service is the reduced extent of the disbecause of tuberculosis, during 1932, were only about one-fourth the number reported for 1922.

A decrease in the number of swine affected with tuberculosis at the time of slaughter also is disclosed by federal meat-inspection records for the same period, compared with previous years. Since most swine are considerably less than a year old when slaughtered, lesions of the disease usually are slight, though sufficient to cause condemnations representing a large aggregate quantity of pork.

The percentage of carcasses more or less affected with tuberculosis during the last 25 years reached its high point in 1924, when 15.2 per cent of the total number of hogs inspected under federal supervision were classed as tuberculous. Advanced tuberculosis was found in 2.7 per cent of the number. During the last fiscal year, the corresponding percentages were 11.4 and 1.59, respectively, a reduction of about 40 per cent in the seriously affected carcasses.

NECROLOGY

CHARLES WALKER SPRINGER

Dr. Chas. W. Springer, of Uniontown, Pa., died July 19, 1932, apparently after he had suffered from a heart attack, while in the swimming-pool at Shady Grove Park, near Connellsville, Pa.

Born at Connellsville, September 12, 1878, Dr. Springer attended local schools and spent his early manhood in the coal business with his father. Later he decided to study veterinary medicine and entered the University of Pennsylvania. Following his graduation in 1905, he spent a year in practice with Dr. George S. Fuller, of Philadelphia. In 1906 he located in Uniontown, where he practiced until his death.

Dr. Springer joined the A. V. M. A. in 1905. He was a member of the Pennsylvania State Veterinary Medical Association and served one term (1923-24) as vice-president, one term (1924-25) as trustee and one term (1929-30) as president. He was a 32nd degree Mason and a Shriner.

Surviving Dr. Springer are his widow, two daughters, one brother and one sister. Burial was at Connellsville.

JAMES J. MULVEHILL

Dr. James J. Mulvehill, of Norwood, Mass., died at the Baker Memorial Hospital, September 17, 1932, after an illness of several months.

Born August 8, 1880, he attended Westwood Grammar School and Dedham High School. He then entered Harvard University for the study of veterinary medicine and was graduated in 1900, before he was of age. He located in Norwood and built up a large practice in the community.

From 1905 to 1908, Dr. Mulvehill was Inspector of Animals, and from 1919 until his death he was agent and inspector of milk and slaughtering for the Board of Health of Norwood. He served Westwood in the same capacity. He was a member of the Massachusetts Veterinary Association and served that organization as president. In 1931, Governor Allen appointed Dr. Mulvehill a member of the Board of Registration in Veterinary

Medicine. He was a past grand knight of Norwood Council, Knights of Columbus, and in 1909 he became the second exalted ruler of the Norwood Lodge of Elks.

Dr. Mulvehill is survived by his widow (née Sophie Mingles), two sons, two brothers and four sisters.

WILLIAM M. STANLEY

Dr. William M. Stanley, of Charles Town, W. Va., died at King's Daughters' Hospital, Martinsburg, W. Va., on September 22, 1932, following an operation for appendicitis.

Born at Halford Bridge, Warwickshire, England, July 24, 1864, Dr. Stanley immigrated to Ontario, Canada, when a young man. Soon afterward he entered the Ontario Veterinary College and upon graduation, in 1895, entered private practice at Hagerstown, Md., remaining there for one year, when he moved to Charles Town.

Dr. Stanley joined the A. V. M. A. in 1927. At the time of his death he was serving as Secretary of the West Virginia State Veterinary Examining Board, which position he had occupied since 1925. He was a member of the West Virginia State Veterinary Medical Association and of the Zion Episcopal Church of Charles Town.

Surviving Dr. Stanley are his widow (née Kate Strider), two daughters and one sister.

H. M. N.

LEO R. HIMMELBERGER

Dr. Leo R. Himmelberger, of Flint, Mich., died suddenly at his home, September 25, 1932, after an illness of about a week, caused by a gastric disorder. He was 44 years of age.

A former resident of Lansing, Mich., and a graduate of the Lansing Central High School, Dr. Himmelberger received three degrees from Michigan State College: Bachelor of Science in Agriculture (1912), Doctor of Veterinary Medicine (1914) and Doctor of Philosophy (1931). For a number of years he was an assistant in the bacteriology laboratory at the Michigan Experiment Station and was largely instrumental in the establishment of the course in medical biology at Michigan State College. Since the establishment of this course he had maintained a deep and helpful interest in the program.

Dr. Himmelberger joined the A. V. M. A. in 1916. At the time of his death, he was pathologist and bacteriologist at Hurley

Hospital, Flint, a position he had held for about ten years. Before going to Flint, Dr. Himmelberger had held positions with the Fort Dodge Serum Company, at Fort Dodge, Iowa, and the Kentucky Agricultural Experiment Station.

Surviving Dr. Himmelberger are his widow, two sons, his father, two sisters and two brothers.

WILLIAM H. CLEMO

Dr. William H. Clemo, of Niles, Mich., died October 3, 1932, after an illness of two weeks.

Born in England, April 23, 1878, Dr. Clemo came to the United States as a boy of eight. His veterinary training was obtained at the Grand Rapids Veterinary College. Following his graduation in 1910, he practiced at various places in Michigan, including Iron Mountain, Eau Claire, Berrien Springs and Niles. He is survived by his widow, one sister and one brother.

HERMAN R. SCHWARZE

Dr. Herman R. Schwarze, of East Saint Louis, Ill., was killed October 25, 1932, when his car struck a culvert at White Heath, near Monticello, Ill. He was on his way to Urbana, to attend the University of Illinois Veterinary Conference, when the accident occurred, and was driving alone, in a very heavy rain.

Dr. Schwarze was a graduate of the Western Veterinary College (1903) and the Chicago Veterinary College (1907). While at the latter institution he was an assistant to the late Dr. Maximilian Herzog, eminent pathologist. He went to the Philippines shortly after graduation and, after several years in the Islands, returned to the United States to accept a position as assistant in the pathological laboratory of the Illinois State Board of Live Stock Commissioners, at Springfield, under Dr. A. T. Peters, who was then in charge.

When the laboratory at Springfield was discontinued, about 1919, Dr. Schwarze was transferred to the Department of Animal Pathology at the University of Illinois, at Urbana. He resigned this position in 1921, to accept one with the United Serum Company, as manager of the Universal Serum Company, at East Saint Louis. January 1, 1923, he became identified with the Corn Belt Serum Company, a connection he retained until his death.

Dr. Schwarze joined the A. V. M. A. in 1913. He was a member of the Illinois State Veterinary Medical Association and secretary of the Illmo Veterinary Medical Association. He was an honorary member of Gamma Chapter of Alpha Psi Fraternity.

EVANS ROBERTS

Dr. Evans Roberts, of Milford, Del., died in the Jefferson Hospital, Philadelphia, October 4, 1932. Death was due to cancer of the stomach. Dr. Roberts was one of two surviving registered non-graduate veterinarians in Delaware. He practiced at Milford for many years and at one time was an executive member of the local Board of Health. He was prominent in church work, Republican politics and business enterprises.

C. C. P.

NORMAN M. PHELPS

Dr. Norman M. Phelps, of Beaver Dam, Wis., died at his home, October 14, 1932, at the age of 76 years. He was a registered, non-graduate practitioner, and the father of Dr. Oscar S. Phelps (Chi. '04), with whom he had been associated in practice for about 25 years. Dr. Phelps served as an alderman in Beaver Dam, several years ago, and was a member of the Dodge County Lodge No. 72, F. & A. M., and the Beaver Dam Chapter No. 26, R. A. M. He is survived by his widow, three sons, one daughter and one brother.

J. O. McC.

PERSONALS

MARRIAGES

Dr. Wallace E. Brown (Colo. '26), of Denver, Colo., to Mrs. Lois Field Meeker, of Santa Fe, N. Mex., August 20, 1932, at Albuquerque, N. Mex.

Dr. S. F. Scheidy (U. P. '29), of Chambersburg, Pa., to Miss Henrietta Kochel, of Reading, Pa., October 1, 1932, at Valley Forge, Pa.

BIRTH

To Dr. and Mrs. L. S. Leider, of Howard, S. Dak., a son, Charles, October 12, 1932.

PERSONALS

Dr. Kermit Schaaf (Mich. '32) recently located at Wawaka, Ind.

Dr. John D. Gadd (U. P. '32) gives a new address: 410 Bosley Ave., Towson, Md.

Dr. George H. Pierce (O. S. U. '09) has resigned as State Veterinarian of Ohio.

Dr. J. H. Krichel (Chi. '16) has removed from Alexis, Ill., to Keokuk, Iowa.

Dr. Paul S. Dodd (Ind. '18) has been reappointed Vermilion County (Ill.) Veterinarian.

DR. DUDLEY D. CONNER (K. C. V. C. '11) has removed from Gulfport, Miss., to Minden, La.

DR. CHAS. W. HUBER (Mich. '32) has opened an office at 114 West High Street, Fostoria, Ohio.

DR. FRANK COLLINS (Chi. '08) reports a change of address from El Paso, Tex., to Monroe, La.

DR. J. P. MACK (N. Y. U. '17) has changed locations, from Leesburg, Fla., to Lake Butler, same state.

DR. RUSSELL E. HALSTEAD (O. S. U. '32) has opened an office for general practice at Xenia, Ohio.

DR. P. H. BLICKENSTAFF (Wash. '23), of Chino, Calif., is attending Ohio State University this fall.

DR. C. C. WARKENTIN (San Fran. '16) has removed from Shafter, Calif., to Crows Landing, Calif.

DR. C. W. TITTLE (Chi. '13) reports a change of address from McAlester, Okla., to Duncan, Okla.

DR. L. R. RICHARDSON (O. S. U. '31) has opened a veterinary hospital at 604 N. Water Street, Kent, Ohio.

DR. F. W. THACKER (Ark. '17), recently of Borgia, Tex., has located for general practice at Carlsbad, N. Mex.

DR. W. E. TRUSSELL (Ont. '32) has taken over the practice of the late Dr. W. M. Stanley, at Charles Town, W. Va.

DR. R. J. BOYLE (McK. '13), of Jo'iet, Ill., has been reëlected Will County Veterinarian by the Board of Supervisors.

Dr. A. C. WALLS (U. P. '93), formerly in the B. A. I. service at National Stock Yards, Ill., is now living at Vero Beach, Fla.

DR. MAX SIEREVELD, JR. (Chi. '09) has been transferred, by the Hartford Live Stock Insurance Company, to San Francisco, Calif.

DR. E. A. RODIER (Wash. '20), formerly of Pullman, Wash., is now at the Veterinary Research Laboratory, Pandacan, Manila, P. I.

DR. M. C. HAWN (Iowa '27), who has been at the North Dakota Agricultural College for several years, is now in Minneapolis, Minn.

DR. R. G. MATTHEW (McGill '97), of the Canada Health of Animals Branch, has been transferred from Hamilton, Ont., to Niagara Falls, Ont.

DR. W. F. BROWNLEE (Chi. '94), of Kirkwood, Ill., was an official veterinarian at the Illinois State Fair, Springfield, the latter part of August.

DR. E. A. HARRIS (Chi. '11) received several painful bruises and a wrenched shoulder in an automobile accident the latter part of September.

DR. C. E. MUMMERT (Ind. '07), of Logansport, Ind., was a judge of horses at a number of county fairs in Indiana, Ohio and Michigan this fall.

DR. W. ED. WELSH (Iowa '27) has resigned his position at Iowa State College and gone to Artesia, N. Mex., with his mother on account of her health.

DR. H. E. VAN DER VEEN (Chi. '17) has been transferred from Genoa City, Wis., to Marengo, Ill. He is with Borden's Farm Products Company of Illinois.

DR. JAMES M. MILLER (McK. '13), of Benton Harbor, Mich., won the Democratic nomination for sheriff of Berrien County, at the September primary election.

DR. JOHN P. HUTTON (O. S. U. '11), of Michigan State College, judged the saddle and light harness horses at the Kalamazoo County (Mich.) Fair in September.

DR. H. E. REA (Ont. '02), of West Branch, Mich., assisted in judging the horses at the Free Stock Show, held at Davison, Mich., recently. About 100 horses were entered.

DR. J. E. WILSON (Colo. '30), formerly with the Los Angeles County Live Stock Department, is now practicing at 1110 Gayley St., Westwood Village, Los Angeles, Calif.

DR. GLEN G. CROSBIE (O. S. U. '31), formerly of Phoenix, Ariz., located at Yuma, Ariz. He is conducting a hospital and boarding kennels at 409 E. Van Buren Street.

DR. HERBERT M. TABBUT (Iowa '32), who was located at Pelican Rapids, Minn., during the summer, has returned to Iowa State College as instructor in veterinary anatomy.

DR. VAN S. JACOBI (McK. '18), of Bunker Hill, Ill., has accepted an appointment in the U. S. Bureau of Animal Industry and has been assigned to meat inspection at Chicago.

DR. WILFORD A. HAYNES (Gr. Rap. '02—Chi. '03), of Jackson, Mich., has been appointed Municipal Meat Inspector, to fill the vacancy caused by the death of Dr. Andrew Campbell recently.

DR. J. P. FOSTER (Ont. '00—McK. '08—Ont. '14), of Minneapolis, Minn., took a 2,400-mile automobile trip through Iowa, Illinois, Indiana, Kentucky and Tennessee during September.

DR. LLOYD D. JONES (Iowa '31) has sold his practice at Hampshire, Ill., to Dr. H. M. Gray (McK. '17), and is now practicing with his father, Dr. F. E. Jones (McK. '99), at Rochelle, Ill.

DR. R. J. HOSKINS (O. S. U. '32) has taken over the hospital and practice of Dr. R. H. Boyd (Ind. '14), of Indianapolis, who has been compelled to give up active work on account of poor health.

DR. MYRON THOM (Wash. '29), for the past three years associated with Dr. C. A. White (Chi. '94), in Los Angeles, Calif., has located at 210 South Raymond Avenue, Pasadena, Calif., for private practice.

DR. GLEN L. DUNLAP (K. S. C. '28) has resigned his position as assistant veterinary pathologist at Massachusetts State College and is now taking graduate work in pathology at the University of Michigan.

DR. E. A. HEWITT (Iowa '17), associate professor of veterinary physiology at Iowa State College, was elected to membership in the American Physiological Society, at the meeting recently held in Philadelphia.

DR. FRED C. CATER (K. C. V. C. '04), who has been in California for several months on account of his health, has become much improved and returned to Sedalia, Mo. He is handling his practice at his old location.

DR. R. A. BRUNSON (K. S. C. '28), who has been associated with his father, Dr. Robert L. Brunson (K. C. V. C. '14), at Corona, Calif., has purchased the practice and home of Dr. P. H. Blickenstaff (Wash. '23), at Chino, Calif.

DR. DANIEL DECAMP (K. S. C. '29) has been transferred from Oregon, Mo., to Fort Wayne, Ind., where he is now working as inspector of dressed poultry, for the Bureau of Agricultural Economics of the U. S. Department of Agriculture.

DR. R. LEARMONTH (Mich. '25), who took postgraduate work at the University of Colorado, last year, has accepted a position on the staff of the University of Pennsylvania School of Veterinary Medicine at Philadelphia. Dr. Learmonth will be associated with Dr. Louis A. Klein (U. P. '97) in work on mastitis.